VIRUS DISEASES AND OTHER DISORDERS WITH VIRUSLIKE SYMPTOMS OF STONE FRUITS IN NORTH AMERICA

Agriculture Handbook 10

UNITED STATES DEPARTMENT OF AGRICULTURE

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VIRUS DISEASES AND OTHER DISORDERS WITH VIRUSLIKE SYMPTOMS OF STONE FRUITS IN NORTH AMERICA[1]

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[1] Project under the Research and Marketing Act of 1946. By action of the council in March 1946 the American Phytopathological Society gave special approval to the handbook as the type of monographic material it favored for publication.

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INTRODUCTION

At the Second Stone Fruit Virus Conference held at Cleveland, Ohio, in 1944 under the chairmanship of V. R. Gardner, a publication committee, consisting of five members, was appointed and requested to revise the text of the Handbook of Virus Diseases of Stone Fruits in North America (*96*).[2] This committee was asked also to include (1) illustrations either colored or black and white for each disease and (2) sections on certain nonvirus and nutritional disorders, the symptoms of which might be confused with those associated with virus diseases.

Stone fruits are necessarily grown in sections affording suitable cultural factors and markets. These sections, especially in western North America, are widely separated geographically. Virus diseases affecting stone fruits occur in all the sections, but they are most prevalent in those where a large number of different kinds of these fruits are associated.

Since some of the diseases are known to have rapid rates of orchard spread, a general policy of carrying on investigations only in the districts of occurrence has been generally followed. The sporadic and wide distribution of the diseases has led to work by a relatively large number of investigators in widely dissociated districts. To bring the results of these studies together, the publication committee adopted the policy of requesting the active cooperation of all investigators known to be working on virus diseases of stone fruits. The plan was to include sections for only diseases which had previously been described in the literature. For each disease a subcommittee consisting of investigators who have been or are actively engaged in research on it was set up. The chairman of each subcommittee, chosen on the basis of original or comprehensive reports, was asked to act as senior author for the section on the disease in question. Except as indicated, associate authors are listed alphabetically without attempt to evaluate the amount of work to their credit. Previously unreported data on some of the diseases are included. In some instances individual responsibility and credit for such data are given by footnotes or directly credited statements in the text. Otherwise each group of authors is responsible for the statements of fact and opinion.

Up to 1930, only 5 virus diseases had been reported on stone fruits and all of these affected peach. Since then, in the short period of approximately 20 years, over 40 new ones have been reported—variously affecting peach, nectarine, plum, sweet cherry, sour cherry, apricot, almond, and many ornamental and wild species of *Prunus*. The discovery of new diseases has been so rapid that factual information concerning many of them is incomplete. Information is particularly short on host range, symptomatology, economic importance, vectors, relative rate of orchard spread, the presence and relationship of virus forms and strains, and other factors important to control.

It is the purpose of this handbook to bring together up-to-date information and to aid recognition of the diseases by including illustrations of diagnostic symptoms. It is believed that the handbook will be useful to students, growers, nurserymen, regulatory officials, and others, and will assist investigators, especially those widely separated geographically, in comparing results and in planning coordinated research. It is hoped that revisions will be made as new information becomes available.

Since virus diseases are still regarded by some as having a certain amount of mystery, it seems necessary to include some general statements about the nature of their causative agents. Viruses are becoming more commonly considered by authorities to be very small efficient parasites, which can live and multiply only in the tissues of their plant or animal hosts. They vary in shape and size: the largest approximate the size of small bacteria and the smallest are generally larger than any of the chemical molecules of their hosts. Some of the common diseases of man caused by viruses are smallpox, mumps, influenza, and measles, and among those of animals are hog cholera, rabies, and foot and mouth disease of cattle.

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[2] Italic numbers in parentheses refer to Literature Cited, p. 252.

In plants, viruses cause diseases characterized by a variety of symptoms. Diseases named "mosaic" from their most prominent symptom are common on many vegetables and weeds and even on shrubs and trees. Other symptoms of virus infection are yellows, leaf curls, excrescences, rosettes, stunts, tissue killing, and even killing of the entire host plant.

In nature, plant viruses are commonly spread by insects, most commonly by those with sucking mouth parts such as aphids, leafhoppers, mealy bugs, and white flies. These insects feed upon plants infected by a virus and take some of the juice containing the virus with them when they migrate to healthy plants, into which they introduce the virus in the process of feeding. Only a few of the vectors are known for virus diseases of stone fruits. Some viruses pass from one host generation to the next through seeds. The only experimental method by which viruses have been transmitted mechanically among fruit trees is some form of grafting.

The incubation period of individual stone-fruit virus diseases varies considerably, depending upon the material and technique. When budding or some other form of grafting is effected in the fall, symptoms do not usually appear before the following growing season. In the case of certain virus diseases, if seedlings or variety trees just breaking dormancy are inoculated with infected tissue, symptoms may appear within a few days. A few virus diseases of stone fruits have long incubation periods of more than a year. Many stone-fruit viruses cause acute, or shock, symptoms and then the host partially recovers and continues to produce only chronic symptoms. For others, symptom production is progressive, and there is neither an acute nor an equilibrium stage.

Since viruses which cause plant diseases are too minute to be seen through an ordinary microscope and cannot be cultured away from living protoplasm, their presence can be recognized only by the characteristic symptoms they produce on their hosts. Thus, the identification of new diseases on the basis of symptoms only, especially if on a single host, may lead to false interpretations. For instance, different viruses may produce similar symptoms on a single host or a single virus may produce dissimilar symptoms on different hosts. Furthermore, a single host may be infected simultaneously by more than one virus, and the symptoms expressed may be the result of the combined effect of all or of the effect of only one, the others being latent. Such combinations are difficult to dissociate. The methods for determining such associations consist principally in the establishment of the existence of components through the use of differential hosts, differential effects of chemical and physical agents, and dissimilar effects with inoculum from a large number of sources.

In some virus diseases there is the complication of the existence of forms or strains of the causal virus capable of producing widely varying symptom expressions. In such cases and until better methods are developed, there is bound to be difficulty in establishing identity and mistakes will be made. It is likely that some of the diseases described in this handbook under separate names will be found to be caused by the same virus or by strains or forms of the same virus. Until exact information is available, most workers follow the practice of describing diseases on new hosts or in new localities as new diseases.

Various systems of naming the causal viruses have been proposed. In most sections of the handbook names according to Johnson's classification (*120*) and those proposed by Smith (*211*), Holmes (*104*, *105*), and McKinney (*145*) are listed. In some sections names according to the Fawcett (*71*) system are also given.

Control is a complex and difficult problem. From a consideration of the large number of diseases described in the handbook, some of which threaten the continued culture of stone fruits in some districts, control may present a dark picture or even appear impossible to some individuals. Without minimizing the difficulties encountered, readers can get encouragement by reflecting on the outlook of peach growers during the height of the peach yellows epidemics, when peach growing in some areas seemed doomed. Today, through unity of effort, peach yellows is no longer considered a major threat to peach growing.

In general, control can be divided into two phases: (1) Prevention by exclusion and (2) reduction by diseased-tree removal or application of methods which reduce the detrimental effects. Individual diseases, often even within limited localities, present their own problems. Before satisfactory control measures can be recommended, the nature of the disease, its distribution, host range, rate and manner of spread, effect on yield, and other facts should be known.

In the past the general recommendation has been to remove virus-affected trees from orchards. For certain diseases and in certain districts this procedure may still be generally recommended. For other diseases there is need for correlated nursery-clean-up and wild-host-removal programs. For certain diseases already generally distributed and for those which have symptomless hosts, which spread rapidly, or which have other characters which make diseased-tree removal impractical, it is necessary to develop other means of control.

Use of resistant or tolerant varieties (varieties which, although infected, are not materially damaged), use of tolerant or resistant rootstock combinations, use of mild-symptom-producing virus forms to protect against infection by more damaging ones, chemotherapy of infected trees, and vector control are approaches which offer promise as control measures. Heat has been used to kill certain viruses in infected budwood and nursery trees, and certain chemicals have reduced infection by others in experimentally inoculated trees. Application of sprays or treatment with chemicals, fertilizers, or other materials has not resulted in the cure of virus-diseased fruit trees in the orchard.

In view of the evidence that certain virus diseases have been and are still being distributed in nursery stock, no control procedure is adequate without provisions to stop such spread. Nurserymen need the assistance of research, regulatory, and extension men and growers in obtaining virus-free scion wood and rootstocks and for establishing procedures to keep the growing nursery stock virus-free. Nursery-improvement programs are under way in various States but vary in procedure and specifications. There is need for rechecking and uniformity in some features to cover provisions for interstate shipment.

Plantsmen should be cognizant of abnormalities which are not caused by infectious agents. For that reason sections describing certain nutritional deficiencies and excesses and other nontransmissible disorders are included. Certain of these are easily remedied by application of the deficient elements. Certain of the bud-perpetuating genetic disorders can be avoided by selecting budwood from unaffected clones.

The report given here is the result of cooperative efforts of the authors of the respective sections and of others, and it could not have been made except for their willingness to pool their information and work together. The report would be incomplete if it were not pointed out that through this cooperative effort workers have been brought in closer contact, and more opportunity for comparison of methods of procedure and results and for exchange of ideas has resulted. It is believed that such association will add much to the solution of the problems presented by virus diseases.

Special credit is due V. R. Gardner, who, as director of the Michigan Agricultural Experiment Station, called the initial conference in 1941 and who acted as chairman of subsequent meetings, to J. R. Magness and G. M. Darrow for suggestions for the manuscript and for assistance in formulating the publication arrangements, and to Annie R. Gravatt and her assistants Lorena Shannon and Eugene Wooden and other members of the Plant Industry Station staff for preparing the handbook for publication.

Credit is also due various plant pathologists who contributed ideas and previously unpublished information and to the following who contributed illustrations for sections of which they are not authors: Harry Andison plate 17, *D;* L. P. Batjer plate 27, *F;* Damon Boynton plate 25, *C, D;* S. R. Cannings plates 17, *B, C,* and 18, *A;* Donald Cation plates 1, *C, D,* 2, *B* to *D,* 24, *B,* and 25, *B,* and figures 1 and 2; L. C. Cochran plates 23, *A, C,* and figure 3; E. S. Degman plate 16, *A;* S. B. Fenne plate 1, *A, B;* W. R. Fisher plate 6, *A, B,* and figure 15; H. L. Garrard plates 23, *B, D,* and 24, *A;* A. Leon Havis plate 24, *C;* Omund Lilleland plate 26, *C;* Roy C. McCue plates 9, *top,* 19, and 22; W. J. Mead plate 1, *E, F;* H. J. O'Reilly plates 10, *B,* and 11, *D;* E. L. Reeves plate 27, *E;* A. O. Simonds plate 8, *B;* Gilbert L. Stout figure 17; Bryce N. Wadley plate 10, *D;* F. B. Wann figures 75 and 76; and J. H. Weinberger plates 2, *A,* and 23 *A, C.*

The cuts for plates 9, 19, and 22 were furnished by the Washington State Department of Agriculture.

The introduction was prepared by the publication committee, composed of Donald Cation, chairman, G. H. Berkeley, L. C. Cochran, F. P. Cullinan, and R. J. Haskell. Reference to the handbook should be made directly to particular authors and sections: for example, Kunkel, L. O., Blake, M. A., and Manns, T. F. Peach Yellows. *In* Virus Diseases and Other Disorders With Viruslike Symptoms of Stone Fruits in North America. U. S. Dept. Agr. Agr. Handb. 10, pp. 1-3, illus. 1951.

VIRUS DISEASES OF PEACH

PEACH YELLOWS

By L. O. Kunkel, M. A. Blake, and T. F. Manns

Other Common Names of the Disease

None.

Names of the Causal Virus

Peach virus 1, Johnson's classification; Prunus virus 1 (Kunkel) Smith; and *Chlorogenus persicae* Holmes.

Geographic Distribution

The disease has been observed from Massachusetts to Virginia on the Atlantic Coastal Plain and southward along the Appalachian Mountains into the Carolinas; westward to Ontario, Canada, and Michigan; and southward to Indiana, Kentucky, and Tennessee. Sporadic outbreaks have been reported in New Hampshire, Iowa, Missouri, Arkansas, Kansas, Texas, and Nebraska, but the disease has never been established west of the Mississippi or in the South.

History and Economic Importance

This first-known peach virus disease (*174, 206, 207, 210*) has resulted in sporadic heavy losses, wiping out entire orchards and even all those in entire peach districts. Great outbreaks occurred in 1791, 1806–7, 1817–21, 1845–58, 1874–78, 1886–88, and 1920. These periods were followed by periods of relative quiescence. The disease has been of minor importance in recent years (*77, 152*). Over most of its range it is now less prevalent than little peach.

Host Range

The following species and varieties are known to be infected: Peach (*Prunus persica*), all varieties; nectarine (*P. persica* var. *nectarina*); apricot (*P. armeniaca*); almond (*P. amygdalus*); Japanese plum (*P. salicina*) varieties Abundance, Chabot, and Satsuma; domestica plum (*P. domestica*); myrobalan plum (*P. cerasifera*); Othello and other ornamental plums (*P. cerasifera* var. *atropurpurea*); and native American plums (*P. hortulana* and *P. americana*). No species of the genus *Prunus* is known to be immune. On myrobalan and Othello plums the symptoms are masked even though the virus is systemic.

Symptoms

Peach.—Fruits on affected peach trees ripen prematurely (pl. 1, *B*), a few days to 3 weeks or more in advance of normal (pl. 1, *A*), and are of inferior quality, usually with a bitter taste (*11*). In varieties that normally develop red coloring in the skin and around the pit the skin is abnormally highly colored, spotted with red and purple, as shown in plate 1, *B*, and the flesh is streaked or marbled crimson with a pronounced red coloring around the pit. The leaves become chlorotic and yellow, fold upward, and tend to roll and droop downward. On typically affected trees thin, wiry, willowy shoots bearing small, narrow, yellow leaves, such as shown in plate 1, *C*, and figure 1, are produced and tend to grow upright from the main limbs. Leaf buds unfold prematurely. In young trees many of the latent buds fail to remain dormant. These unfold into yellowish leaves, scarcely an inch in length, giving the tree a bushy appearance. In advanced cases the ends of the old limbs die and twigs bearing small yellow leaves are produced from limbs and trunks. Affected trees normally die in 2 to 6 years, but those affected with mild strains of yellows may live for a much longer period, particularly if given good cultural treatment.

On greenhouse trees the earliest symptom is the clearing of the veins of the tip leaves (*130*). Young leaves at the tips of infected branches stand straight if healthy and bend over, or are sickle-shaped, if diseased. Production of thin, upright-growing shoots bearing small, slightly chlorotic leaves is a conspicuous

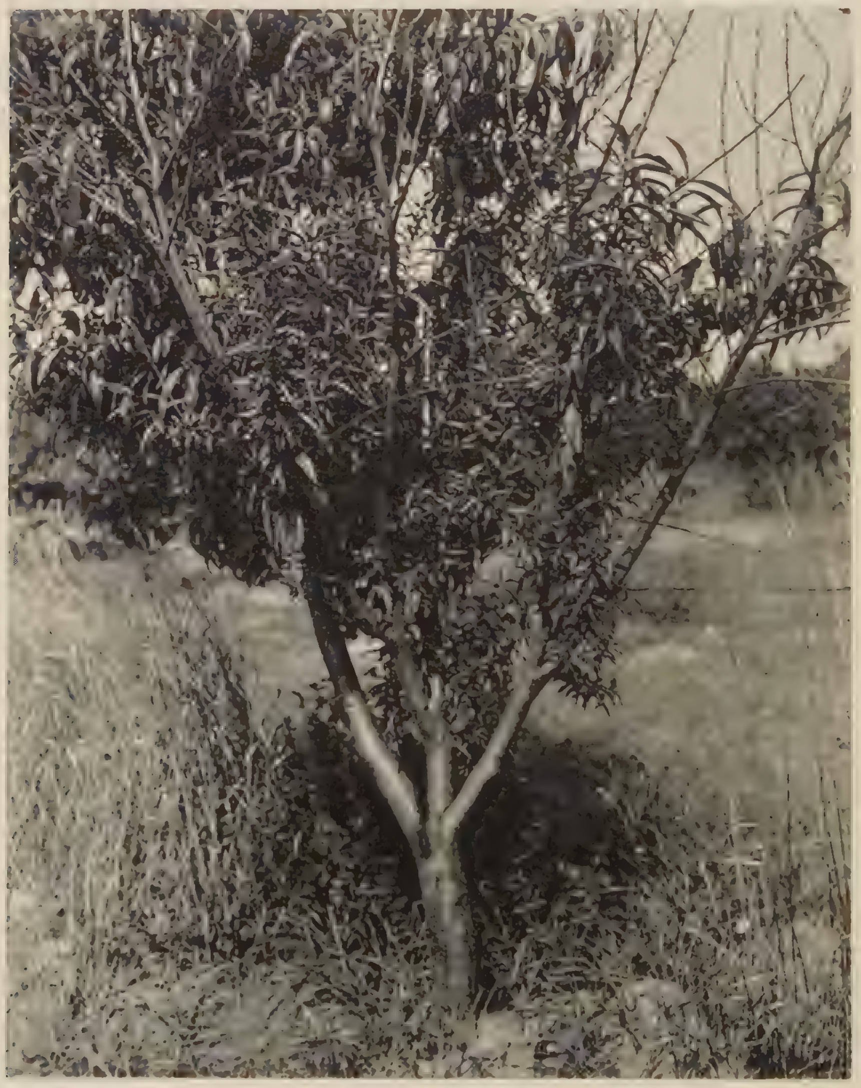

Figure 1.—Peach tree affected with peach yellows (advanced stage), showing clusters of wiry shoots growing from main arms.

symptom of yellows after the disease is well established. Young trees frequently live 1 or 2 years after infection.

Apricot and other hosts.— On apricot, almond, American and hortulan plums, David peach (*P. davidiana*), almond cherry (*P. glandulosa*), and Manchu cherry (*P. tomentosa*) symptoms are similar to those on peach and about as severe. On Abundance plum, German Prune (*P. domestica*), wild-goose plum (*P. munsoniana*), and Japanese apricot (*P. mume*) symptoms are similar to but milder than those on peach. On myrobalan and Othello plums symptoms are very mild or lacking. On Wickson plum, a hybrid resembling *P. simonii*, symptoms are similar to but more severe than those on peach.

Diagnostic or Unusual Characteristics

Production of slender, upright-growing shoots by affected trees makes peach yellows easy to diagnose. The disease does not persist in warm peach-growing districts of the South, probably because of inactivation of the virus by heat (*131*).

Transmission

Transmission by budding and grafting has been demonstrated repeatedly (*147*). Kunkel (*129*) demonstrated that the plum leafhopper (*Macropsis trimaculata* (Fitch)) transmits yellows; this was confirmed by Hartzell (*79, 80*), Manns (*149*), Manns and Davies (*151*), and Manns and Manns (*152*). Kunkel listed negative results for 14 species of sucking insects. Hartzell failed to get transmission with 47 species of insects and mites, using 20 species in significant numbers. Manns listed negative results with several species. A relatively small percentage of positive transmissions were obtained by the 3 investigators.

Incubation Period

Under orchard conditions the incubation period may be as long as 3 years. Under appropriate greenhouse conditions it may be as short as 40 days. Peach yellows virus travels much more rapidly downward than upward; hence, infections produced near the tip of a tree cause symptoms much earlier than infections occurring at or near the ground level (*128*). Kunkel (*133*) showed that a bud-contact period of 8 to 11 days is necessary for virus transmission.

Control Measures

Care in using only healthy material for propagation and eradication of diseased trees in orchards as soon as symptoms appear have proved effective control measures. On the basis of findings by Manns and Manns (*153*) that certain infected plum trees lived for many years and harbored large numbers of the plum leafhopper, it is suggested that plums not be planted in the vicinity of peach orchards. Spraying for control of the plum leafhopper is also suggested.

Remarks

Peach yellows under the present methods of control does not seem to be a serious disease in well-kept commercial orchards except in isolated instances.

LITTLE PEACH

By T. F. Manns, M. A. Blake, and L. O. Kunkel

Other Common Names of the Disease

Peach littles.

Names of the Causal Virus

Chlorogenus persicae var. *micropersica* Holmes; little peach virus; peach virus 3, Johnson's classification; Prunus virus 1A Smith; and little peach strain of peach yellows virus.

Geographic Distribution

Little peach occurs in eastern United States as far south as North Carolina and in Ontario, Canada (*43, 44*). It is found in approximately the same area as peach yellows.

Economic Importance

Little peach has superseded peach yellows in importance in most districts in recent years, but in some districts yellows is as potent as ever. It is the most destructive virus disease of peach in the northern peach-growing districts east of the Mississippi except in Connecticut, Massachusetts, and New York, where X-disease is now present.

Host Range and Varietal Susceptibility

The host range of little peach (*5, 43, 148, 150, 153*) is similar to that of yellows. Peach (*Prunus persica*), plums, and apricot (*P. armeniaca*) are affected. Manns (*150*) showed that certain varieties of Japanese plum (*P. salicina*), Abundance, Chalco, and Chabot, are symptomless carriers; other varieties, such as Satsuma and Santa Rosa, and crosses such as Simon may show symptoms, but live for many years. Myrobalan plum (*P. cerasifera*), which is used extensively as a rootstock, is a symptomless carrier. All the domestica plums and prunes (*P. domestica*), as well as the wildgoose plum (*P. munsoniana*), are susceptible.

Symptoms

On young seedling peach trees the first symptom is a distortion of the young leaves at the tips of affected branches. Production of an abnormally large number of short branches along the main stem, especially near the soil level, is common. These branches are more upright than normal, but not as vertical as those affected with yellows. Shortening of internodes and general stunting of the tree result. Newly infected trees are generally of a deeper green color than is normal for healthy trees. After the disease becomes chronic, the mature leaves turn slightly yellow and twiggy growth develops along the branches (pl. 1, *D*). After 2 or 3 years a smaller number of new shoots are produced.

On older peach trees the symptoms are at first most prominent on a single branch or portion of a tree. The foliage is frequently of a darker green color and appears more compact and bushy than normal. The compact appearance is due partly to shortened internodes and partly to the increased production of leaves on short, lateral branches or spurs on the 2- and 3-year-old wood. The crowded leaves tend to droop, become leathery, bend inward, and curl toward the branches on which they originate, giving a compact, bushy, and drooping appearance (fig. 2). An unusual number of short, spurlike shoots frequently sprout from the bases of the larger limbs. As the disease becomes chronic, the leaves turn lighter green to yellow. As the disease weakens the trees, the compact, bushy character of the growth becomes less evident. The fruits on affected trees or branches (pls. 1, *E*, and 2, *C*) are smaller and ripen several days to 3 weeks later than normal (pls. 1, *F*, and 2, *B*). They generally have an insipid flavor. The pits are reduced in size, and the kernels are either undeveloped or fail to germinate.

On susceptible plum and apricot varieties the symptoms are similar to those on peach, but usually much milder.

Diagnostic or Unusual Characteristics

The foliage of diseased trees in the earlier stages is darker than that of healthy ones and has a heavy leathery appearance. Blake (*10*) did not regard a darker green in early stages of little peach as of diagnostic value. The fruits usually mature several days or more later than is normal and are smaller. Manns has observed that where Japanese beetles (*Popillia japonica* Newm.) are abundant trees affected with peach yellows and little peach are infested by thousands of the insects, whereas the healthy trees attract few.

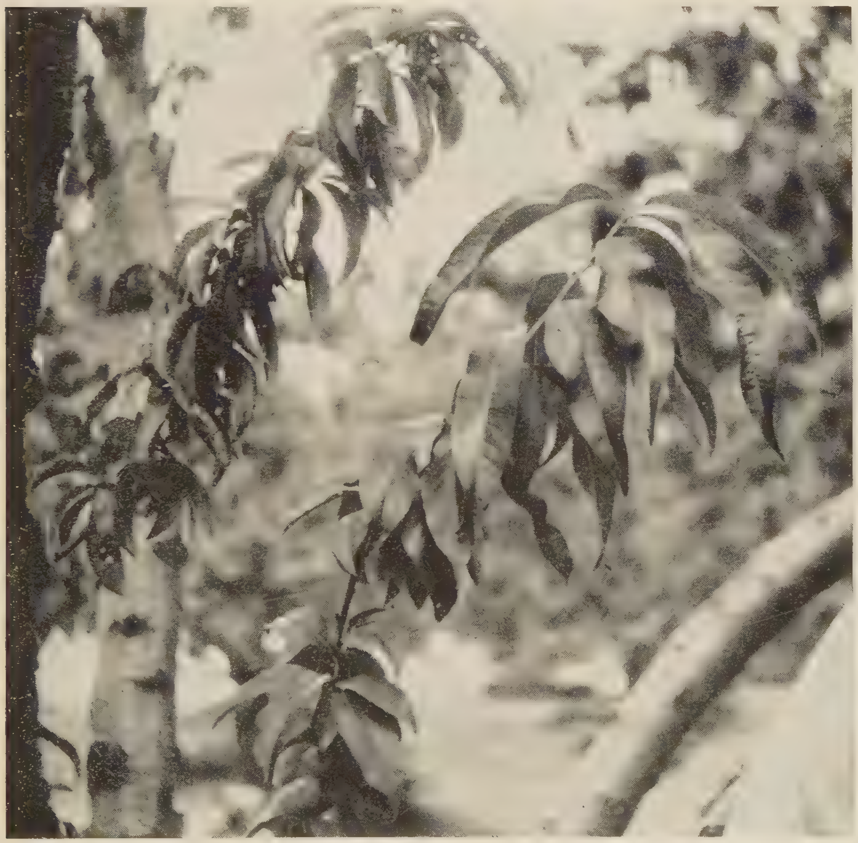

Figure 2.—Peach shoot showing drooping, clustered foliage, a symptom characteristic of little peach, in contrast with unaffected shoot at right.

Transmission

Transmission is effected by budding or grafting of the root or top with infected tissue and by means of the plum leafhopper (*134, 148, 150, 153*).

Incubation Period

The incubation period (*133, 150*) in the field may vary from 1 to 2 years after budding with infected tissue, but in the greenhouse it may be less than 2 months under optimum conditions.

Thermal Relationships

The little peach virus is inactivated by heat treatment at the same temperature and exposure that proved effective for the control of peach yellows (*131*).

Immunologic Relationships

Kunkel (*130, 131*) showed that peach trees having little peach virus are immune from infection by peach yellows virus and that likewise trees having yellows virus are immune from infection by little peach virus, but that neither of the causal viruses of these diseases gave any protection against peach rosette virus. Manns (*150*) reported that when healthy bearing trees are budded simultaneously near the growing points with one bud infected with peach yellows virus and another with little peach virus, the yellows symptoms will be the first to show, but that very shortly afterward the little peach symptoms will appear and be the dominating ones. However, Kunkel (*133*) showed that, when peach seedlings are simultaneously inoculated with peach yellows and little peach viruses by budding, the bud placed in the top position determines the nature of the acquired disease.

Blake questioned whether it is correct to say that trees having little peach virus are immune from yellows, he and associates having occasionally found trees which had one branch with a distinct case of yellows and another with a distinct case of little peach (*11*). On one branch the fruits prematured, while on another they were immature.

Control Measures

Diseased trees should be removed as soon as discovered. Removal or indexing of suspected masked carriers (plums and certain other species of *Prunus*) and the use of contact sprays to eliminate the insect vector (*Macropsis trimaculata* (Fitch)) are suggested (*150*).

Remarks

Little peach is spread by the same vector, is inactivated by similar heat treatment, and has a similar host range and approximately the same geographical distribution as peach yellows (*134, 148, 150, 153*). Little peach virus also protects a plant against infection by peach yellows virus. For these reasons, the little peach and peach yellows viruses are considered to be closely related and strains of the same virus.

PEACH ROSETTE

By J. A. McClintock, L. O. Kunkel, and H. H. Thornberry

Other Common Names of the Disease

None.

Names of the Causal Virus

Peach rosette virus; peach virus 2, Johnson's classification; Prunus virus 2 (McClintock) Smith; *Nanus rosettae* Holmes; and *Carpophthora rosettae* Holmes.

History and Geographic Distribution

Peach rosette was first noted in Georgia about 1881 (*207, 208*). Subsequently the disease was reported in Alabama, South Carolina, Tennessee, West Virginia, Missouri, and Oklahoma (*143*). It has been reported also in Kentucky, Illinois, Indiana, Arkansas, and Kansas.

Economic Importance

Peach rosette has caused serious losses locally in peach orchards, but it is of minor importance in comparison with peach yellows, little peach, phony, and peach mosaic. It could become of greater importance if established in native stands of *Prunus pumila* bordering peach orchards in the Great Lakes area.

Host Range and Varietal Susceptibility

Peach rosette virus causes symptoms on peach (*P. persica*); apricot (*P. armeniaca*) varieties Moorpark, Royal, and Wilson; almond (*P. amygdalus*); mazzard cherry (*P. avium*); sour cherry (*P. cerasus*) seedlings; chickasaw plum (*P. angustifolia*); Japanese plum (*P. salicina*) varieties and hybrids Kelsey, Ogon (Botan), Maynard, and Red June; damson plum (*P. insititia*); and sand cherry (*P. pumila*). Natural infections have been recorded on peach and chickasaw plum in southern United States.

Thornberry (*237*) reported that Wilson apricot approaches the category of a symptomless carrier. Marianna plum, a hybrid variety used principally as a stock, was shown by McClintock (*143*) to be immune from infection by the peach rosette virus.

Tobacco (*Nicotiana glutinosa*), periwinkle (*Vinca rosea*), and tomato (*Lycopersicon esculentum*) were infected by transmission of peach rosette virus from affected peach trees through dodder (*Cuscuta campestris* Yuncker) (*135*).

Symptoms

Peach.—Peach-rosette-affected trees (fig. 3) are more striking in appearance than trees affected with peach yellows. The disease is more rapid in progress than peach yellows. Symptoms generally appear with the recurrence of growth in the spring. The first-formed leaves are normal in size for the variety, but commonly they fold inward or arch backward. The leaves usually turn yellow in contrast with the various shades of green in healthy leaves. On these first-formed leaves red spots develop, and usually the leaves fall in early summer. As new terminal growth has extremely short internodes, the newly formed leaves are closely appressed into distinct rosettes (pl. 2, *A*). The older of these leaves are progressively shed, leaving only tufts of younger leaves near the tips of naked twigs.

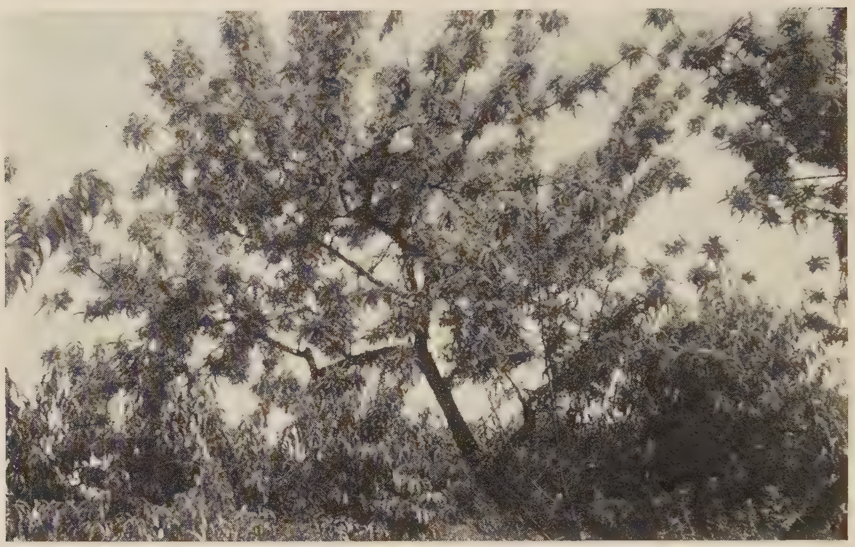

***Figure 3*.—Elberta peach tree affected with peach rosette, showing contrast with a normal branch at far left.**

Trees of bearing age usually produce blossoms, but they do not set and mature fruits on branches which show leaf symptoms. In the majority of cases peach-rosette-affected peach trees die within the year in which symptoms first appear, but exceptions to this were observed by McClintock (*143*) in peaches growing on peach-rosette-immune Marianna plum stock.

Plum.—On chickasaw plum rosette symptoms are much less striking than on peach. Shortening of the internodes is less pronounced, and usually there is less premature defoliation of infected plums. On this species of plum the virus appears to be less virulent; therefore infected wild plums may survive for more than a year after symptoms become identifiable. From these observations McClintock concluded that the wild chickasaw plum and related southern species or hybrids are the native hosts of peach rosette virus. Infected wild plums in proximity to orchards are suspected of being the major source of inoculum for cultivated stone fruits. On damson plum symptoms consist of rosetted growth and mottled leaves. Affected Maynard plum trees are stunted, and the growth is rosetted.

Apricot.—On affected Royal apricot the shoots are weak and have shortened internodes, but they are not rosetted as severely as affected peach. The growth is stunted, and the leaves are mottled. On Moorpark apricot the growth is stunted, and the leaves are mottled greenish yellow (*143*). On Wilson apricot the symptoms are extremely mild. There is a tendency for witches'-brooms to form and for the number of shoots from main branches to increase. The leaves are not mottled but are slightly yellowed, and the petioles turn reddish. The trees may show some stunting of growth, especially when they are infected when small. Otherwise, Wilson apricot approaches the category of a symptomless carrier (*237*).

Other hosts.—On affected almond the growth is stunted and the leaves are yellowish green and rosetted.

On mazzard cherry affected with peach rosette the growth is markedly reduced, and the leaves are smaller and more rolled than normal ones during the second season. The leaves are yellowish green, and the growth is tufted as on affected peach. The terminal buds fail to become dormant. Resistance was indicated in mazzard, as one affected tree remained alive for 23 months after the appearance of the symptoms.

Symptoms resembling those of peach rosette were seen on seedlings of sour cherry growing near infected peach (*236*).

On affected sand cherry there was a witches'-broom growth of the new shoots and the leaves were smaller and lighter green than those of healthy plants, but affected plants were not killed.

On periwinkle peach rosette virus causes rosettes similar to those on peach. It causes wilting and death of rapidly growing tomato and tobacco.

Diagnostic or Unusual Characteristics

The quick death of infected peach trees makes peach rosette on this host partially self-eradicating (*243*). Evidence to date indicates that apricots and wild and cultivated plums are more resistant to the lethal effects than are peaches. Although peach rosette is of southern habitat, the symptoms produced on peach by artificial inoculation in Michigan were characteristic of those reported in the South.[3] These findings, coupled with the northward progress of natural infections, indicate that this disease could become of importance in northern orchards.

Transmission

Natural spread from infected wild plums to cultivated plums and peach and from diseased to healthy trees in orchards is apparent, but no vector is known. Attempted transmissions with sap and insects were negative (*144*). Budding or grafting of tops or roots of peach and plums transmits the virus provided tissue union results between diseased and healthy plants (*209*). The virus was apparently transmitted through dodder from peach trees to periwinkle, tomato, and tobacco. Serial transmission in these species has been effected by grafting. The virus has not been transmitted from herbaceous hosts to peach by grafting or through dodder.

Incubation Period

The first appearance of symptoms varies under field conditions, depending on the season of the year and the point of inoculation. Kunkel (*133*) determined that a bud-contact period of 8 to 14 days is necessary for virus transmission from peach to peach.

Thermal Relationships

Kunkel (*131*) found peach rosette somewhat more refractory to heat treatment than little peach, red suture, and peach yellows. Peach rosette virus in bud sticks was destroyed by 8- and 10-minute treatments at 50° C. It was not destroyed in buds exposed as long as 2 hours at 40°.

Control Measures

Systematic eradication of infected trees in orchards and of native wild *Prunus* growing near orchards as soon as symptoms are observed has proved effective and is recommended. If diseased trees are allowed to remain in the orchard, peach rosette has been observed to increase rapidly and reach epidemic proportions, affecting whole orchards in 3 to 5 years. Even under these conditions spread can be reduced to a few trees within 1 or 2 years by prompt diseased-tree removal in the spring.

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[3] Cation, D. Correspondence with the authors.

Remarks

Kunkel (*130*) showed that peach yellows and little peach produce no inhibiting effect in peach against infection with peach rosette virus. Determination of the natural host range and the vectors would contribute to the control measures that could be recommended.

RED SUTURE

By Donald Cation and C. W. Bennett

Other Common Names of the Disease

None.

Names of the Causal Virus

Red suture virus; Prunus virus 4 (Bennett) Smith; peach virus 5, Johnson's classification; and *Chlorogenus persicae* var. *vulgaris* Holmes.

History and Geographic Distribution

Red suture occurs in Michigan and Maryland (*5, 244*). Trees inoculated with the virus from Maryland and Michigan showed that the virus from both sources produced the same disease. A few suspected cases were reported from southern Indiana. There is evidence that red suture was present in Michigan as early as 1911 (*230*), but thus far it has not been reported with certainty from any other peach-growing section except Maryland, where it probably was introduced on infected nursery stock.

Economic Importance

Red suture on peach is responsible for annual losses of up to 3 percent of the trees in districts where it is established, and periodic losses in epidemic years involve large portions of entire orchards. On the whole, red suture is of less importance than little peach in Michigan.

Host Range and Varietal Susceptibility

Red suture is known to occur only on peach (*Prunus persica*) and Abundance variety of Japanese plum (*P. salicina*). Results of graft-inoculation tests made by Cation indicate that sweet and sour cherries (*P. avium* and *P. cerasus*) are immune. No immune varieties of peach have been found.

Symptoms

Peach.—Symptoms of red suture are found on both the fruits and the foliage (*5, 44*). Infection also produces a characteristic type of tree growth. The fruits ripen several days prematurely, particularly on the suture side, which softens and may be fully mature while the other side is still green and hard. The suture side is frequently, but not always, swollen or bulged (pl. 2, *E*). On the side that ripens early, the flesh is rather coarse, stringy, and exceptionally watery. Many of the fruits have a rough, bumpy contour, more pronounced on the suture side. The early-ripening side of red-colored fruits is splashed or blotched with an abnormally dark red to purple, the color being more pronounced on the apexes of the bumps. Yellow-colored fruits, such as those of Gold Drop, are not reddened but are deeper yellow on the suture side. The flavor of the fruits is usually, but not always, insipid. The suture side tends to bruise or break down in shipment, resulting in a loss in marketing.

Trees with red suture are generally off-color as compared with adjacent normal trees. A diseased tree as a whole presents a yellowish-green to greenish-bronze appearance, but if heavily fertilized with nitrogen it may have a green color approaching normal. The difference in color of diseased and healthy trees is more noticeable at certain periods in the season, particularly several weeks after petal fall and also just prior to fruit maturity. Diseased trees usually take on autumnal coloration prematurely. In diseased trees many vigorous 2-year-old branches show a characteristic type of growth. On such branches buds develop and grow into short spurlike outgrowths with a resulting crowding and clustering of the leaves along the branch (pl. 2, *D*). On similar branches of a normal tree some of the buds remain dormant, a few grow long shoots, and only a few develop into spurlike growths. A tree that has had the disease for several years shows less dense interior growth than normal. Diseased trees may live for 8 years or more.

Plum.—On the variety Abundance, Cation found that symptoms are indistinguishable from those caused by peach yellows. Affected trees have a lighter color than normal, and the fruits ripen a little early. The varieties Red June (*P. salicina*) and Wickson (*P. salicina* × *P. simonii*) show severe symptoms when affected with peach yellows, but these varieties were little affected with the red suture virus.

Diagnostic or Unusual Characteristics

Peach trees with red suture are very similar to those with little peach in type of tree growth and in foliage characteristics. Red suture is diagnosed most readily from fruit symptoms and is distinguished from little peach by a preponderance of bulged sutures and early, uneven ripening of the fruit. Diseased trees are most easily detected in orchards receiving good culture.

Peach trees inoculated with both red suture and rosette mosaic viruses or with red suture and sour cherry yellows viruses will show rosette mosaic or sour cherry yellows, respectively, the following season, according to Cation. Peach trees grafted concurrently with buds infected with red suture and peach yellows viruses will show the symptoms of the disease represented by the uppermost bud.

Transmission

Transmission has been accomplished by grafting with bark tissue, with or without buds, from diseased trees. Graft inoculations with leaf or fruit tissue have not been attempted. The results of inoculations by means of the plum leafhopper (*Macropsis trimaculata* (Fitch)) have been negative, but there were too few of them to be conclusive.

Incubation Period

In the field the symptoms on a mature tree appear the second year, or 22 months after inoculation. Symptoms are not apparent on 1-year-old nursery trees grown from diseased buds and frequently not in the second year of growth.

Thermal Relationships

Kunkel (*131*) found that the red suture virus is inactivated by heat, particularly when bud sticks are immersed in a water bath at 50° C. for 3 minutes. The thermal-inactivation point is very close to that of the peach yellows and little peach viruses.

Control Measures

In most places red suture has been kept in check by periodic orchard inspection and removal of diseased trees, but in a few localities it is endemic. The nursery practice of indiscriminate purchase of propagating material from growers should be discouraged. Indirect evidence indicates that such practice was responsible for the spread of the disease from Michigan to Maryland. Preferably bud sticks should be taken from inspected trees or from those in orchards containing no disease. Heat treatments could be used to insure freedom from disease if the propagation wood is questionable. Nursery inspection is of little value, since symptoms are not evident on young trees.

Remarks

On the basis of disease symptoms and the thermal-inactivation point of the causal virus (*131*), red suture appears to be related to peach yellows and little peach. It seems probable that the red suture virus may have arisen from the peach yellows or the little peach virus by mutation. The restricted distribution of red suture indicates that, if the virus is a mutant in the peach yellows group, such mutants occur rarely.

ROSETTE MOSAIC

By Donald Cation

Name of the Disease

The disease was first described on peach and was called "rosette mosaic," a name which refers to two of the outstanding symptoms.

Name of the Causal Virus

No Latin binomial has been proposed, and for convenience the virus will be referred to as rosette mosaic virus.

History and Geographic Distribution

The disease has been present in Berrien County, Mich., at least since 1917 (*45, 50*) and has been found subsequently in a few scattered orchards in that county. A few cases have also been reported in New York (*82*).

Economic Importance

Rosette mosaic seriously affects production of individual trees, but because of the relatively few infected trees in Michigan it has not been of great importance. The disease is locally serious in Eau Claire Township, Berrien County, Mich., where there was a considerable increase in incidence in 1947.

Host Range and Varietal Susceptibility

So far as known the virus of rosette mosaic is limited to peach (*Prunus persica*) and plums. All varieties and seedlings of peach tested proved susceptible, the severity of reaction varying with the variety. The following varieties of peach are listed in the order of the severity of effect of the disease: South Haven, Halehaven, J. H. Hale, Elberta, Carman, Golden Jubilee, Kalhaven, Ambergem, and Gold Drop. Damson plum (*P. insititia*) and Burbank, Red June, and Abundance plums (*P. salicina*) appear to be symptomless carriers, but Italian Prune (*P. domestica*) and Wickson (*P. salicina* × *P. simonii*) plums express symptoms.

Symptoms

Peach.—The symptoms on peach may consist of delayed foliation; chlorotic mottling of early-formed leaves, generally accompanied by distortion (fig. *4, A*); and a shortening of internodes, which frequently crowds the leaves into a so-called rosette. These symptoms are somewhat variable because there are forms of the virus, and the peach varieties respond differentially.

When chlorosis of the leaves occurs, it is evident early in the season. Chlorotic areas vary in size and shape and range from yellow or cream color to translucent. Tissue growth in these chlorotic areas is retarded, and distortion results. Less definite retardation gives a wavy, or undulating, leaf margin.

Leaves formed later in the season on affected trees appear more normal in size and shape, but actually they are about two-thirds as wide as leaves on unaffected trees. Diseased trees have a rosetted appearance due to the crowding of leaves on twigs with shortened internodes. Affected trees have a darker green color than normal.

Premature defoliation is not common. A variable number of normal branches may be interspersed with affected ones on an infected tree (pl. 3, *E*).

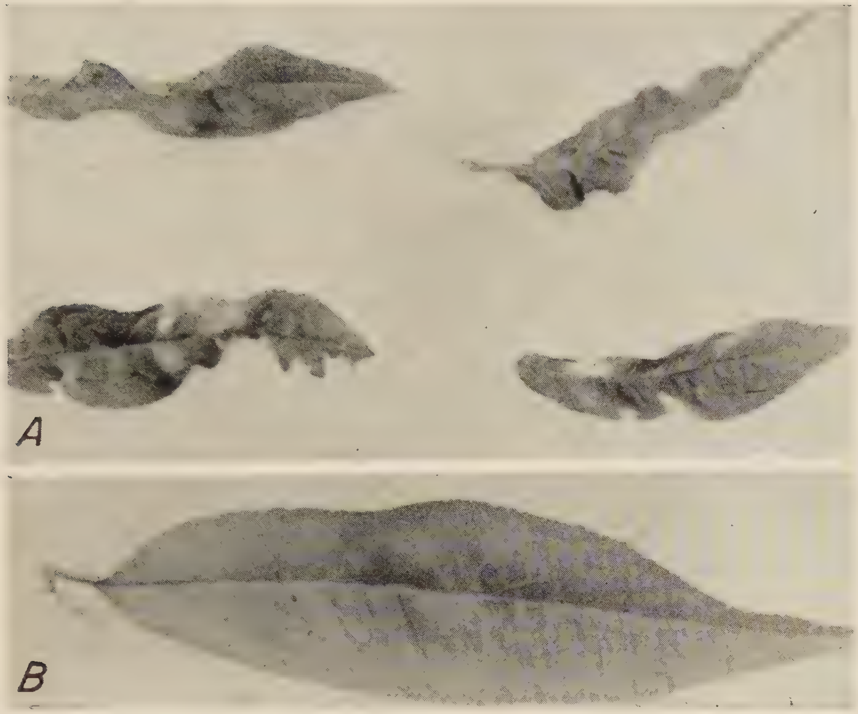

Figure 4.—*A*, **Misshapen leaves with chlorotic areas from peach tree affected with rosette mosaic; *B*, comparable normal leaf.**

Inoculation with buds from such normal-appearing branches did not result in transmission.

Plum.—The Italian Prune variety of plum inoculated with rosette mosaic virus showed symptoms simulating those of prune dwarf (*85*). Infected trees varied in response according to the form of the virus concerned, some having strap-shaped leaves and others leaves that were only slightly dwarfed, roughened, and thickened. Wickson plum was slow to express symptoms but had smaller leaves and definite rosetting in the third and fourth years after inoculation. Damson plums did not express definite symptoms, but it is perhaps significant that the nonvigorous trees in one orchard were the only ones shown to be carrying the virus.

Diagnostic or Unusual Characteristics

The symptoms of rosette mosaic on peach have some similarities to those of prune dwarf (*85*), sour cherry yellows, and peach mosaic (*114*). Peach is more severely affected by rosette mosaic than by prune dwarf, whereas Italian Prune is less markedly affected by rosette mosaic and more severely affected by prune dwarf. Rosette mosaic virus does not produce the dieback or ring spot symptoms characteristic of the sour cherry yellows virus complex on peach. In two cases of attempted transmission rosette mosaic virus did not cause sour cherry yellows symptoms on Montmorency sour cherry. Sour cherry yellows symptoms on peach are generally distributed throughout the tree the year after bud inoculation, whereas the symptoms of rosette mosaic tend to be localized in a few branches during the first season. Rosette mosaic has not resulted in color breaking of blossoms, in distortion of fruit, in shot holes or emargination of leaves, or in the marked and general chlorosis frequently caused by peach mosaic virus (*114*).

Transmission

Transmission of the disease has been effected by grafting, by the transfer of soil from diseased to healthy trees, or by planting healthy trees in a location previously occupied by diseased trees. Transmission by means of soil has not precluded the possibility of transfer by insect vectors, particularly the black peach aphid (*Anuraphis persicae-niger* (Smith)). Some 30 attempts to transmit the virus by means of such aphids, however, did not result in disease expression.

Incubation Period

The incubation period has varied from a few weeks under optimum conditions in the greenhouse to 9 months and even as long as 21 months in the field. The length of time required depends apparently on the virulence of the virus, the susceptibility of the variety, and the state of tree growth during the incubation period. When transmission with contaminated soil was started by July 15, symptoms appeared 10 months later. Trees planted in diseased soil in the spring showed symptoms the following spring, but trees planted in the fall did not show symptoms until the second spring.

On seedlings 18 to 24 inches tall the shortest incubation period was 12 to 16 days when rapid-transmission techniques were used (*86*). With the defoliation technique 14 to 19 days was required for symptom expression and with the shading technique 18 to 21 days (*97*).

Thermal Relationships

Hildebrand[4] was unable to inactivate rosette mosaic virus by soaking the bud sticks in a hot-water bath at 50° C. for 20 to 22 minutes.

Control Measures

Removal of diseased trees and avoiding replanting for several years after their removal are indicated control measures. Peaches should not be planted immediately after plums unless the plums are known to be free from rosette mosaic. Plum varieties should be indexed for freedom from virus before being used as propagating stock by nurserymen.

Remarks

A relationship between the causal viruses of rosette mosaic and prune dwarf is indicated by the similarity of symptoms on peach and Italian Prune and by their closely similar thermal relationships.

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[4] Hildebrand, E. M. Correspondence with the author, 1948.

PHONY

By Lee M. Hutchins, L. C. Cochran, and William F. Turner

Names of the Disease

"Phony disease" and "phony peach disease" are the names that have been used in recent literature. In Georgia, where the disease was first discovered, the names "pony peach" and "collar oedema" are sometimes used by growers. "Phony" appears to be an adequate name for this well-known disease and is employed in the present discussion.

Names of the Causal Virus

The following names have been applied to the virus of phony: "Phony peach virus"; "peach virus 4," Johnson's classification; "Prunus virus 3 (Hutchins) Smith"; and "*Nanus mirabilis* Holmes." Under the Fawcett system of virus nomenclature the virus might be named "*Prunuvir mirabilis*." Pending adoption of an international system of virus nomenclature, the authors prefer to use the name "phony virus."

History and Geographic Distribution

The first-known observation of phony was made on two peach trees at Marshallville, Ga., about 1885, by Samuel Rumph, who discovered and introduced the Elberta peach variety. The dwarfed specimens were called "pony trees" by Rumph and were regarded as a curiosity. Their number increased, and by 1915 thousands of peach trees were known to be affected in a limited district in central Georgia. Illustrated papers calling attention to and describing the disease appeared in 1920 (*167*) and 1928 (*107*).

Research into the cause of phony was begun in 1921 by the Bureau of Plant Industry, United States Department of Agriculture. A fairly exhaustive, well-illustrated account of the research and other information acquired was published in 1933 (*112*)[5] and is freely used in the present discussion. Some of the more important information may be found in other publications (*109, 110*).

In 1928 phony was definitely attributed to an infective virus (*108*). This finding was announced at a Federal quarantine hearing in Washington, D. C., in the fall of that year, at which time the disease was known to have spread over a large district in central Georgia and a smaller district in eastern Alabama. As a result of the hearing, Federal Quarantine No. 67 was promulgated, effective June 1, 1929 (*241*); this restricted movement of peach and nectarine nursery stock and peach roots within and from affected districts. Also in 1929 a Federal-State cooperative program for systematic survey and removal of phony-diseased peach trees was inaugurated.

By 1932 about 35 million peach orchard trees had been inspected. The disease had been found in all the South Atlantic States below Virginia, in all the Gulf States, in Tennessee and Arkansas, and in a few locations in the southern parts of Oklahoma, Missouri, and Illinois (*238*, *253*). Subsequent surveys, from 1933 to 1947, extended the district of known occurrence of the disease in most of the States named and discovered the disease in Indiana, Kentucky,

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[5] Publication now out of print.

Maryland, and Pennsylvania. However, apparently the disease has now been eradicated from these last four States and from some other States in the lightly affected area, as indicated under Control Measures.

These extensive annual surveys established the general area of distribution of phony as being in southeastern United States. The disease has not been positively reported elsewhere in this country or abroad.

Economic Importance

Phony is first in importance among virus diseases of peach in Georgia and some others of the Southern States. About 1,657,877 phony-diseased trees have been removed by control agencies since 1929.[6] Previous to that time it was estimated that over a million orchard trees had been ruined by the disease in central Georgia.

Phony does not cause early death of the affected trees; but it induces marked dwarfing of new growth and of fruits, and this results in a smaller crop of peaches, most of which are undersized. The longer the tree has the disease, the more pronounced this effect becomes. The bulk of the fruit from phony trees falls in the smaller commercial grades or is graded out entirely. Trees that contract the disease before the bearing age never come into profitable bearing, and mature trees that have had the disease several years produce no commercial crop of fruit.

Previous to the eradication campaign instituted in 1929, the potential destructiveness of phony was determined in several central-Georgia orchards, where the incidence of diseased trees was recorded annually on orchard maps. Where the disease was very prevalent and spread was rapid, it was not uncommon for half of the trees in an orchard to show the disease at the age of 6 to 8 years and for 99 percent to be affected at the age of 12 years. Under such conditions the orchards are ruined at an age when they should be most productive.

Host Range

So far as known, phony virus is limited to the genus *Prunus*. Knowledge of species susceptibility is incomplete, but the tests and observations reported here have been made. Susceptible to natural infection are all varieties, races, and hybrids of peach (*P. persica*); peach trees propagated on rootstocks of other species, such as almond (*P. amygdalus*), apricot (*P. armeniaca*), Japanese apricot (*P. mume*), David peach (*P. davidiana*), and hortulan plum (*P. hortulana*); and seedlings of apricot, Japanese apricot, chickasaw plum (*P. angustifolia*), and Mexican plum (*P. mexicana*) (*117*). Trees of all the above-named species on their own roots are also susceptible to artificial inoculation through root grafting with root pieces from phony-diseased peach trees. Also, typical phony symptoms are produced on peach following root-graft inoculations from affected trees of the other species; the peach has been used in this way as a test plant for verifying the presence of phony virus in the other species.

Although no truly symptomless carriers of phony virus have been found, the species that tend to produce bushy growth, such as chickasaw plum, are much less strikingly affected by the disease than are those species that normally produce vigorous, elongated branches. Where known diseased trees are growing beside known normal trees of such species, differences in growth characters and seasonal response may be clearly evident, whereas in thickets of wild seedlings visual identification of trees carrying the virus may be extremely difficult and highly uncertain.

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[6] U. S. Bur. Ent. and Plant Quar., Div. Dom. Plant Quar. Phony Peach and Peach Mosaic Control Project Annual Reports. [Processed, unpublished.]

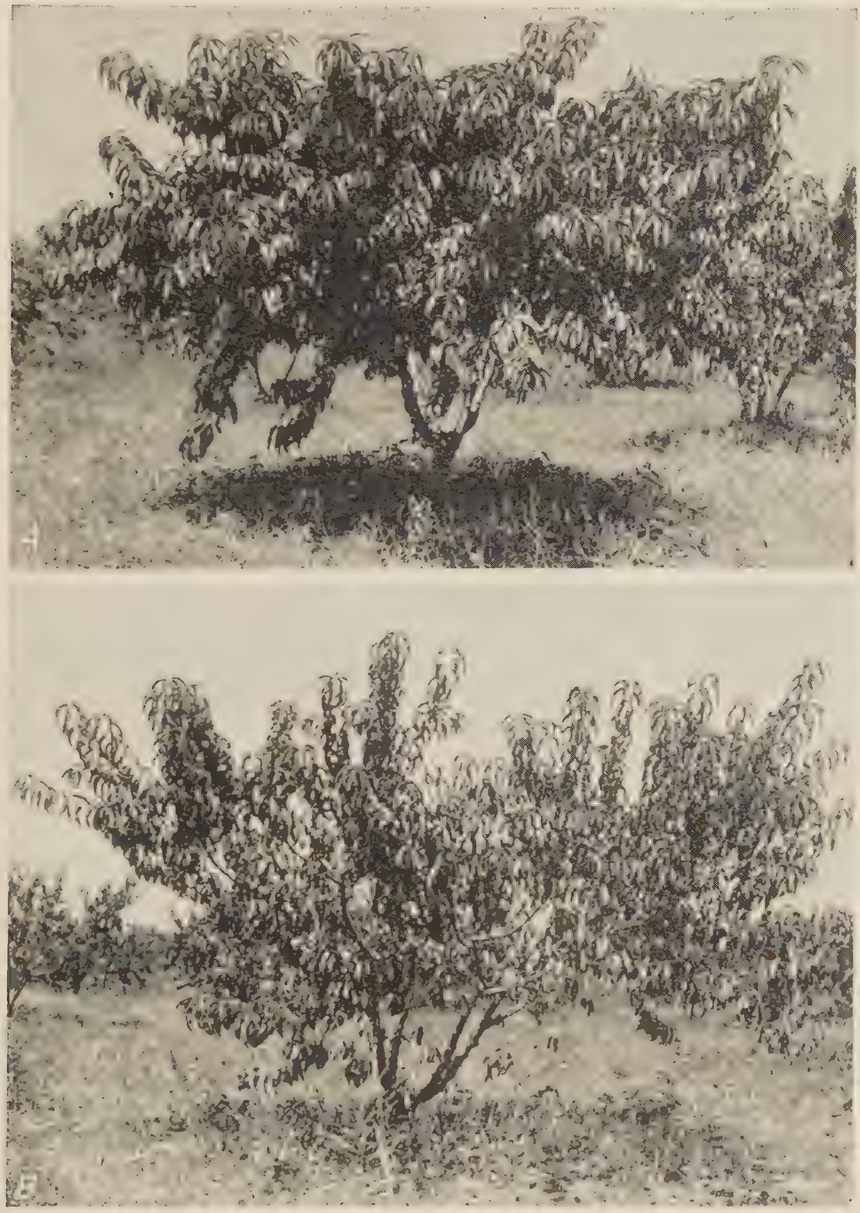

Figure 5.—*A*, Six-year-old Elberta peach tree affected with phony, showing the dense shadow cast by the heavy, appressed foliage, Fort Valley, Ga. *B*, Comparable normal tree in same orchard.

Symptoms

The symptoms of phony are most striking when the trees are in full foliage and in good vigor. With the onset of symptoms, the phony tree develops shortened internodes, rather profuse lateral branches, and flattened dark-green leaves, giving the appearance of compact, dense growth with luxuriant foliage. Decided dwarfing of new growth results, and the periphery of the upper crown tends to take on a uniform, rounded appearance (fig. 5). Phony trees may live many years. However, after several years of the disease, the wood appears brittle and there may be a notable dying back of the terminal twigs and branches. Vigorous, new terminal growth cannot be forced. As large limbs die or are broken down, they are not replaced and the tree takes on a ragged appearance. Even on such trees the foliage tends to be characteristic of the disease; that is, it is greener and flatter than that of normal trees. In spring, phony trees flower and leaf out earlier than normal ones of the same variety, and they hold their foliage later in the fall (fig. 6).

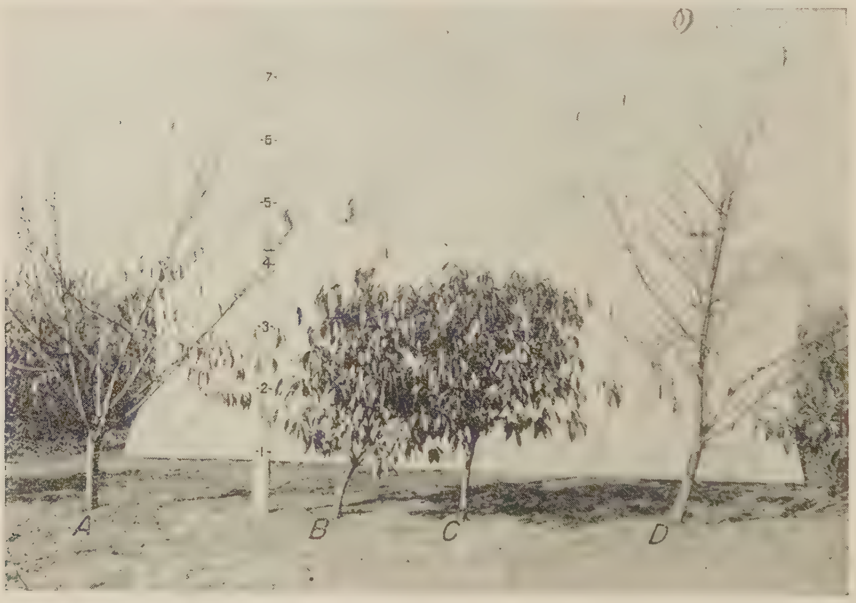

Figure 6.—**Four nursery peach trees of same age, photographed October 24, 1931, Fort Valley, Ga.: *A* and *D*, Normal trees; *B* and *C*, trees affected with phony as a result of a root-graft inoculation, showing dwarfing and retention of foliage.**

The fruits on phony trees are well formed and ripen a few days earlier than normal fruits, but they are reduced both in size and in number per tree (fig. 7; pl. 3, *A*, *B*). They may be somewhat poorer in flavor than normal fruits, but they are generally more highly colored. Seeds from phony peaches average smaller than normal seeds, but they give excellent germination and produce normal seedlings.

Infection with phony virus does not induce spindling growth or rosetting, does not cause yellowing, rolling, or spotting of leaves, and does not produce lesions, galls, or hypertrophies.

Identification of the disease in the affected tree is made from the aspects of

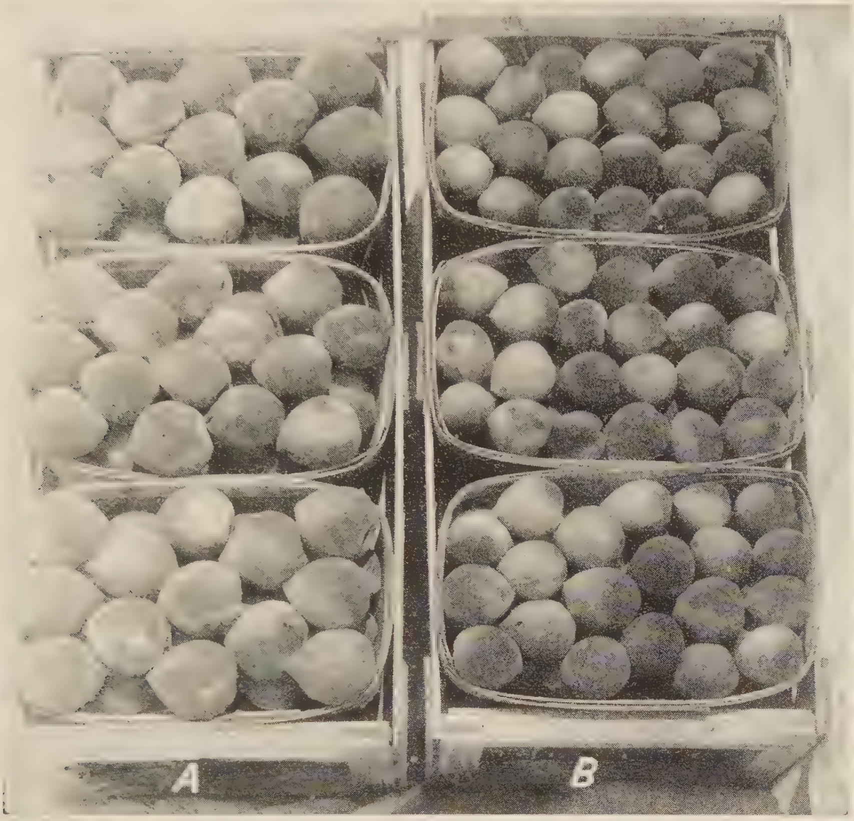

Figure 7.—*A*, **Commercial pack of best-grade (1⅞- to 2¼-inch diameter) Hiley peaches from normal tree; *B*, similar pack of 1½- to 1⅞-inch-diameter fruits from a tree affected with phony.**

the tree as a whole and should not be attempted from detached specimens. Phony trees are most easily identified in cultivated orchards, where comparison with vigorous normal trees of the same age and variety can be made. Identification of the disease in wild seedlings and in orchards of mixed varieties may be difficult unless the symptoms are very pronounced. Pruning, fertilizing, and cultivating in keeping with good horticultural practice will induce vigor and growth that intensify symptoms and bring out strong contrasts between the diseased and the normal trees.

All of the many peach varieties observed in the States where phony occurs are susceptible to the disease, and there are no significant differences in symptom manifestation. In *Prunus* species that tend to produce bushy growth, such as *P. angustifolia,* it has been possible to identify phony in artificially inoculated trees by comparing them with normal trees of the same species, where the two were growing side by side in nursery rows; but in thickets of wild seedlings visual identification in place may be unsatisfactory. In such cases, if identification is important, root inoculation of peach nursery test trees should be resorted to.

Chemical Identification Test

A useful confirmatory aid to identification of phony is provided by a simple and rapid chemical test, easily applied in laboratory or field (*112*).

The test reagent is prepared by acidulating 100 cc. of absolute methyl alcohol with 15 to 20 drops of concentrated, chemically pure hydrochloric acid. The degree of acidity required varies somewhat with the source of the alcohol and with the specimens. Absolute ethyl alcohol may be used, but it may require higher acidulation and for a rapid test must be hot.

For the test, select unblemished, whole-root sections 4 to 6 inches long, with a woody cylinder 0.5 to 0.75 inch in diameter, and remote from malformations or injuries in the root branches from which the specimens are taken. Wash the specimens, remove excess water, and cut entire transverse sections of woody cylinder 0.5 to 1 mm. thick and immerse them in a few cubic centimeters of the reagent in a flat container. Before the test the wood should be white or nearly so, fresh, and in good condition throughout. If numerous, well-distributed purplish spots up to 2 mm. in diameter show up against a background of whitish or faintly purplish wood after a few minutes in the reagent, a positive test for phony is indicated (pl. 3, *C, D*). Parallel check tests should be run on known normal roots. At the end of the test period for phony root sections, the normal sections should still be free from color or should show a fairly even purple tint with no tendency to localization in well-distributed, intensely purple spots. If acidulation is too strong or if the sections are allowed to remain too long in the reagent, the entire surface of the sections (either normal or phony) may display a purple color.

In well-developed cases of phony it is usual for each suitable root to give a good positive test throughout the year. In old cases of the disease, brown flecks may occur in the wood. These will usually turn purple in the reagent, and numerous additional purple spots will develop during the test. Only the wood gives the test; the bark shows no reaction. In some cases phony stem wood may show a positive reaction to the test, but it is much less satisfactory than root wood.

The test as just described applies to the peach. Other *Prunus* species may give a somewhat variable reaction, the test being excellent in some, as in *P. davidiana,* for example, and less striking in others.

Diagnostic or Unusual Characteristics

Several characteristics of phony are seldom encountered in other known virus diseases of peach or other woody plants. The chemical test (pl. 3, *D*) is specific for phony and is a valuable confirmatory aid to identification. Localization of the virus in the woody cylinder is very unusual and has been reported in only one other case, namely, Pierce's disease of grape (*106*) in California. Phony appears to be the only virus disease of stone fruits in which symptom manifestation in the shoot, regardless of whether or not the virus is present there, develops only after the symptomless root system is thoroughly invaded by the virus; the incubation period is about 18 months. Techniques for proving suspected virus diseases of woody plants now should include use of root grafts and of other graft inoculum containing living wood, especially when transmission is not secured by bark or bud grafts.

Transmission and Natural Spread

The early work to determine whether the disease is infectious was confined to the peach. In extensive trials over several years the disease was not transmitted by grafting scions or buds from diseased trees on normal trees, but it was transmitted when a diseased tree and a normal tree were united by a root graft or when a whole-root section from a diseased tree was grafted on a root of a normal tree. It was concluded from these results that the virus is confined to the root system of the diseased tree. New data [7] indicate that phony virus occasionally exists in the tops or portions of the tops of affected peach and plum trees.

When patches of root bark from diseased trees were grafted on the root or the root collar of normal trees, the virus was not transmitted, whereas it was invariably transmitted by root graftage when the inoculum consisted of living bark and wood. In the peach, therefore, the virus is apparently localized in the woody cylinder (*113*). Attempts to transmit the disease by juice from affected roots were unsuccessful, and transmission by root grafting was accomplished only when there was definite growth union. The theory was advanced that for transmission of the virus by grafting it was requisite that a bridge of new wood be formed to connect the woody cylinder of the inoculum with that of the tree undergoing inoculation.

Four species of Cicadellidae have recently been reported as vectors of phony under experimental conditions (*240*). These species are *Homalodisca triquetra* (F.), *Oncometopia undata* (F.), *Graphocephala versuta* (Say), and *Cuerna costalis* (F.). All are members of the subfamily Tettigellinae. The four species, when feeding on peach, appear to limit their attentions to the twigs and small branches, usually to 1- and 2-year-old growth. These observations indicate that the virus is present at times in some portions of the tops of the trees. Such information as is available on the feeding habits of the subfamily to which these vectors belong is not at variance with the data suggesting a localization of the virus in the woody cylinder.

In the light of these discoveries, which furnish valuable information important to the control procedures, renewed and extensive investigations combining pathological, entomological, and horticultural aspects of the problem are being carried out. A large part of this work is located at the United States Horticultural Field Laboratory, Fort Valley, Ga.

Incubation Period

The precise length of the incubation period of phony under conditions of natural infection is not known. However, in young orchards planted with known normal trees and surrounded by a heavy incidence of disease in older adjacent orchards, none of the young trees develop the disease the first year. An occasional tree, perhaps 1 in 200, may develop the disease at the end of the second growing season; in 1 exceptional case the number exceeded 3 percent. Under such favorable conditions for natural spread, ordinarily 3 to 5 percent of the trees may develop the disease during the third growing season after planting.

The incubation period of the disease in peach nursery or orchard trees inoculated by root-piece grafts in late winter is ordinarily about 18 months. Multiple inoculations of large trees may shorten the incubation period by a few months. When scions of peach and other species are grafted in the tops of phony-diseased trees, the growth from the scion develops typical symptoms at once. When dormant peach nursery trees are grafted by the taproot to large roots severed from phony-diseased trees but otherwise undisturbed in the soil, the new growth from the nursery tree will display phony characters immediately.

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[7] Hutchins, L. M., Cochran, L. C., Turner, W. F., and Weinberger, J. H. Recovery of phony virus from the tops of certain affected peach and plum trees. (Manuscript in preparation.)

Symptoms of phony on the shoots of peach are apparently induced directly by effects of the virus in the roots. Shoot growth from virus-bearing scions grafted on healthy trees appears normal during the first year of growth but usually becomes phony along with the remainder of the tree during the second year. Until the major portion of the root system is invaded by the virus, the symptom-provoking conditions apparently are not sufficiently developed to induce symptoms and terminate the incubation period. As soon as symptoms appear in the shoot, all root pieces taken from a diseased tree will transmit the disease, whereas a few months before the termination of the incubation period many of the root pieces will not transmit the disease.

Thermal Relationships

Phony virus can be inactivated in dormant peach nursery trees 1 year after root-graft inoculation by immersing the trees for 40 minutes in water maintained at 48° C. (*116*).

Control Measures

After the discovery in 1928 that phony is caused by an infective virus, control activities were instituted through quarantine regulations and a program for eradication of diseased trees.

Effective June 1, 1929, Federal Quarantine No. 67 (*241*) forbade the movement of peach and nectarine trees or peach roots of any variety of tree or shrub grafted on such roots from regulated districts to any other part of the United States except under a special permit from the United States Department of Agriculture. Such permits were issued only if the nursery and its environs for a radius of 1 mile had been inspected by Federal or State inspectors and no phony had been found within the locality for at least 2 years. The Federal quarantine was revoked in 1934. At present the movement of susceptible nursery stock is controlled by uniform State quarantines. The requirement for certification is that any phony trees found within the 1-mile environs of the nursery must be eradicated prior to June 30 of the current year. As a result of intensive control activities, phony has been eradicated from the districts producing most of the peach nursery stock in the Southeast.

Phony peach eradication was originated by the Bureau of Plant Industry in 1929, the work being carried out in cooperation with State agencies and property owners. The initial activities established the approximate boundaries of the whole affected area, and the districts of heavy, general, moderate, and light infestation. The program of orchard inspection and diseased-tree removal that was inaugurated soon resulted in a marked decrease in the total number of phony trees (*238, 239*).

In 1934, in keeping with a general plan of reorganization that was effected at that time, direction of the phony peach disease control project was transferred from the Bureau of Plant Industry to the Bureau of Entomology and Plant Quarantine, United States Department of Agriculture. Activities expanded in succeeding years with the aid of special allotments and increased State cooperation. Progress of the work is extensively treated in a series of annual reports [8] and in special publications (*52, 74, 75, 238*). Phony has now been found in 17 States, 10 of which have been much less heavily infected than the others. By 1944 no phony had been found during three or more consecutive years in Indiana, Kentucky, Maryland, North Carolina, Oklahoma, Pennsylvania, and Illinois. In Mississippi, Tennessee, Texas, Missouri, Louisiana, Arkansas, and South Carolina the incidence of diseased trees was drastically reduced from a total of 13,415 affected trees in 1936 to a total of 1,724 affected trees in 1946, and the disease has apparently been eliminated from a large number of counties.

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[8] See footnote 6, p. 18.

After initial decrease in some districts, however, further reductions proved more difficult. Several factors served to handicap the control activities rather severely. Among these are the long incubation period and the lack of knowledge as to the time of year when natural spread takes place most rapidly. It may well be that until eradication of diseased trees can be supplemented by other means of control, the most that can be hoped for in some of the rapid-spread districts is a maintenance of status quo.

The work is continuing under an enlarged program as a portion of a project entitled "Phony Peach and Peach Mosaic Control" under the general direction of the Division of Domestic Plant Quarantines, Bureau of Entomology and Plant Quarantine, United States Department of Agriculture, Gulfport, Miss. [9]

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[9] The project leader, A. E. Cavanagh, maintains headquarters at Room 523, Federal Building, San Antonio, Tex. Local offices are maintained in several other States.

PEACH MOSAIC

By Lee M. Hutchins, E. W. Bodine, L. C. Cochran, and Gilbert L. Stout

Name of the Disease

The name "peach mosaic" was applied to the disease in 1932 by Hutchins (*111*), because of the similarity of its symptoms to those of mosaics well known on other plants. At that time this name served to distinguish the disease from the other virus diseases affecting peach, and it was not foreseen that other distinct diseases of the mosaic group would be found on stone fruits. Since "peach mosaic" is now recognized as the common name of this specific disease, others of the group in which mosaic is incorporated in the name should be distinguished by qualifying adjectives or by new names of which mosaic is not a part.

Names of the Causal Virus

The virus causing peach mosaic has been variously named. The chronology of the names is as follows: Peach virus 6, Johnson's classification; Prunus virus 5 (Hutchins) Smith; *Marmor persicae* Holmes; and *Flavimacula persicae* (Holmes) McKinney. Under the Fawcett system the name might be "*Prunivir flavimacula*." Until more complete agreement is attained, it is the preference of the authors to refer to the causal virus by adding the term "virus" to the common name of the disease; thus, "peach mosaic virus."

History and Geographic Distribution

In July 1931 W. F. Turner[10] called Hutchins' attention (*114*) to a new disease on peach at Bangs, Tex., subsequently named "peach mosaic." At about the same time Bodine (*30, 31*), observed seven affected trees near Palisade, Colo. Extensive surveys (*227*)[11] by State and Federal agencies have shown that the disease exists in southern California, southern Utah, Arizona, New Mexico, southern Oklahoma, western Arkansas, western Colorado, Texas, and Mexico.

The widespread existence of the disease in the Rio Grande Valley is evidence that it had been present there for a long time. It is plausible that the causal virus could have been carried from there in nursery stock, especially in wild plums, in which hosts symptoms are usually absent, to sections where peach varieties, cultural practices, and environment made its presence more evident. Both the pottawattamie, or wildgoose, plum (*Prunus munsoniana*) and the American plum (*P. americana*) are common in the Rio Grande Valley, where they have been planted along ditchbanks. The fruits are prized for jelly, and plants have been moved as sucker shoots from one locality to another. Index tests performed by budding onto peach have shown that a high percentage of the wildgoose-type plums in certain localities and occasional trees of the American plum are infected.

Economic Importance

The loss from reduction in quality and quantity of fruit due to the effects of the peach mosaic virus is sufficient to make culture of peach varieties as severely damaged as J. H. Hale, Elberta, and Rio Oso Gem unprofitable. Others of the freestone group, particularly those having J. H. Hale or Elberta heritage, also are severely damaged. Some varieties, including most of the clingstones and several of the freestones, even though mosaic variably reduces their yields, are sufficiently tolerant to produce profitable crops. Such varieties when carrying severe symptom-producing forms of the causal virus are hazardous to less tolerant varieties in mixed or adjacent plantings.

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[10] In charge of the phony peach eradication campaign, U. S. Bureau of Plant Industry.

[11] U. S. Bur. Ent. and Plant Quar., Div. Dom. Plant Quar. Phony Peach and Peach Mosaic Control Project Annual Reports. [Processed, unpublished.]

Damage from the disease is nearly negligible on plums, prunes, apricots, and almonds in southern California, but little is known of its effects on these hosts in sections with other climates. In cooler sections fruits on some affected apricot trees were observed to be very bumpy and worthless. The fruits on certain almond trees were bumpy, but there was no reduction in yield or quality of the nuts.

Host Range

Extensive studies indicate that the host range of the peach mosaic virus is limited not only to the genus *Prunus* but also to certain species and even to certain horticultural varieties within the genus. Further specification is indicated by the fact that individual horticultural plum varieties were found susceptible to one form of the virus but immune from others. The cherry and cherrylike species generally appear to be immune. The following species have been experimentally infected: Peach (*P. persica*); nectarine (*P. persica* var. *nectarina*); almond (*P. amygdalus*); apricot (*P. armeniaca*); domestica plum (*P. domestica*); Japanese plum (*P. salicina*); Japanese apricot (*P. mume*); David peach (*P. davidiana*); damson plum (*P. insititia*); American plum (*P. americana*); chickasaw plum (*P. angustifolia*); wildgoose, or pottawattamie, plum (*P. munsoniana*); myrobalan plum (*P. cerasifera*); Mexican plum (*P. mexicana*); Bessey cherry (*P. besseyi*); tangut almond (*P. tangutica*); and Manchu cherry (*P. tomentosa*). All attempts to infect sweet cherry (*P. avium*) varieties growing on mazzard or mahaleb rootstock and mazzard (*P. avium*) seedlings, mahaleb cherry (*P. mahaleb*), sour cherry (*P. cerasus*) growing on mahaleb rootstock, western chokecherry (*P. virginiana* var. *demissa*), desert apricot (*P. fremontii*), desert almond (*P. fasciculata*), and hollyleaf cherry (*P. ilicifolia*) have failed.

The peach mosaic virus has been recovered from many naturally infected horticultural varieties of peach including flowering types. It has also been recovered from naturally infected French (Agen) prune, several varieties of apricot, and a few varieties of Japanese plum growing in orchard formation. The virus has been recovered from naturally infected wild pottawattamie (wildgoose), chickasaw, American, Mexican, and myrobalan plums; Japanese apricot, David peach, tangut almond, Bokar plum (*P. bokhariensis*), hortulan plum (*P. hortulana*), flatwoods plum (*P. umbellata*), and hog plum (*P. reverchonii*). Recovery from Japanese plums has been rare and limited to certain varieties such as Maynard (*36*), Becky Smith, Flaming Delicious, and Cain Seedling. Attempts at experimental infection indicate that some varieties of both Japanese- and domestica-type plums are variably affected with or immune from some or all of the forms of the peach mosaic virus. Chickasaw plum is an important host reservoir of the virus in Texas, where it exists in extensive thickets and has been found generally infected, but without symptoms, in the vicinity of mosaic-infected peach orchards. The pottawattamie plum, likewise, has been found to be a common reservoir of the virus in New Mexico, Arizona, and Utah, where it has been planted along irrigation canals and fences. Escape seedlings of cultivated plums forming thickets along fences and irrigation canals in New Mexico have been found generally infected.

Symptoms

Peach.—Symptomatology of peach mosaic is made complex by the presence of many causal virus forms (*32, 35, 60, 61, 115*), some of which produce symptoms so mild that diagnosis is difficult and others of which produce ruinous effects on peach varieties like J. H. Hale. Given enough sources of the virus, a gradient in symptoms can be produced (*57*). Further, the disease has been observed to proceed in a sequence of acute followed by chronic phases in which infected trees show strong symptoms and later appear to recover partially (*56*).

On J. H. Hale peach trees inoculated the previous fall and, similarly, on orchard trees showing first-year symptoms, spring growth is variously retarded;

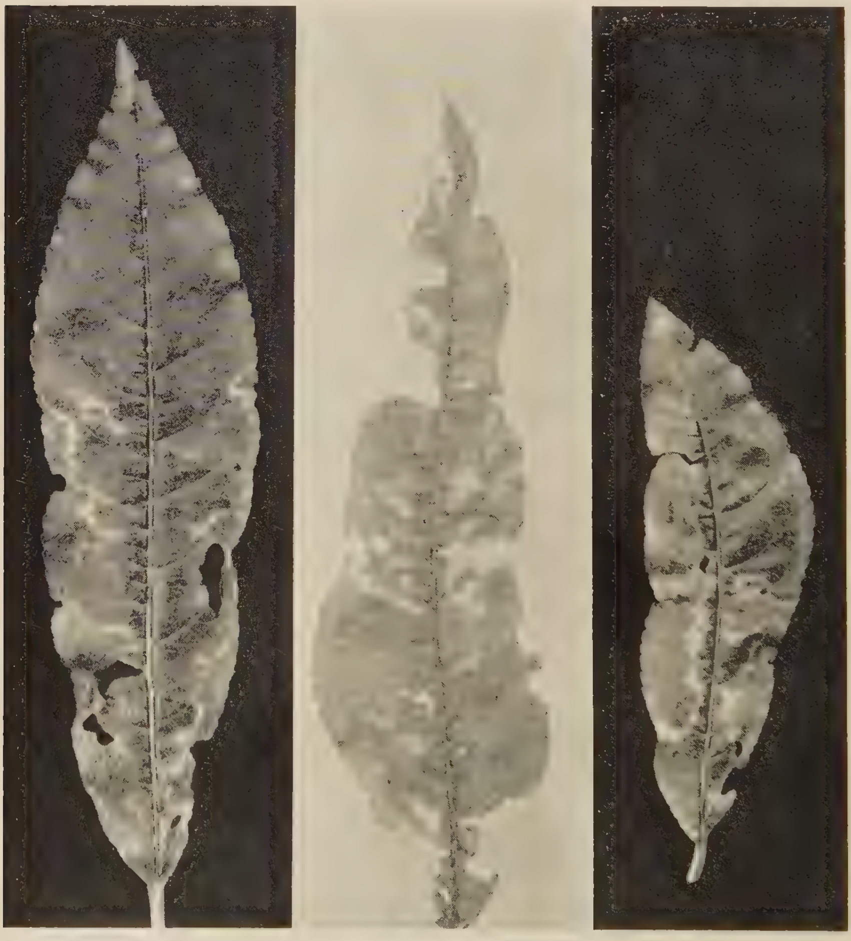

Figure 8.—Leaves of Early Wheeler peach, a partially tolerant variety, showing summer symptoms of peach mosaic.

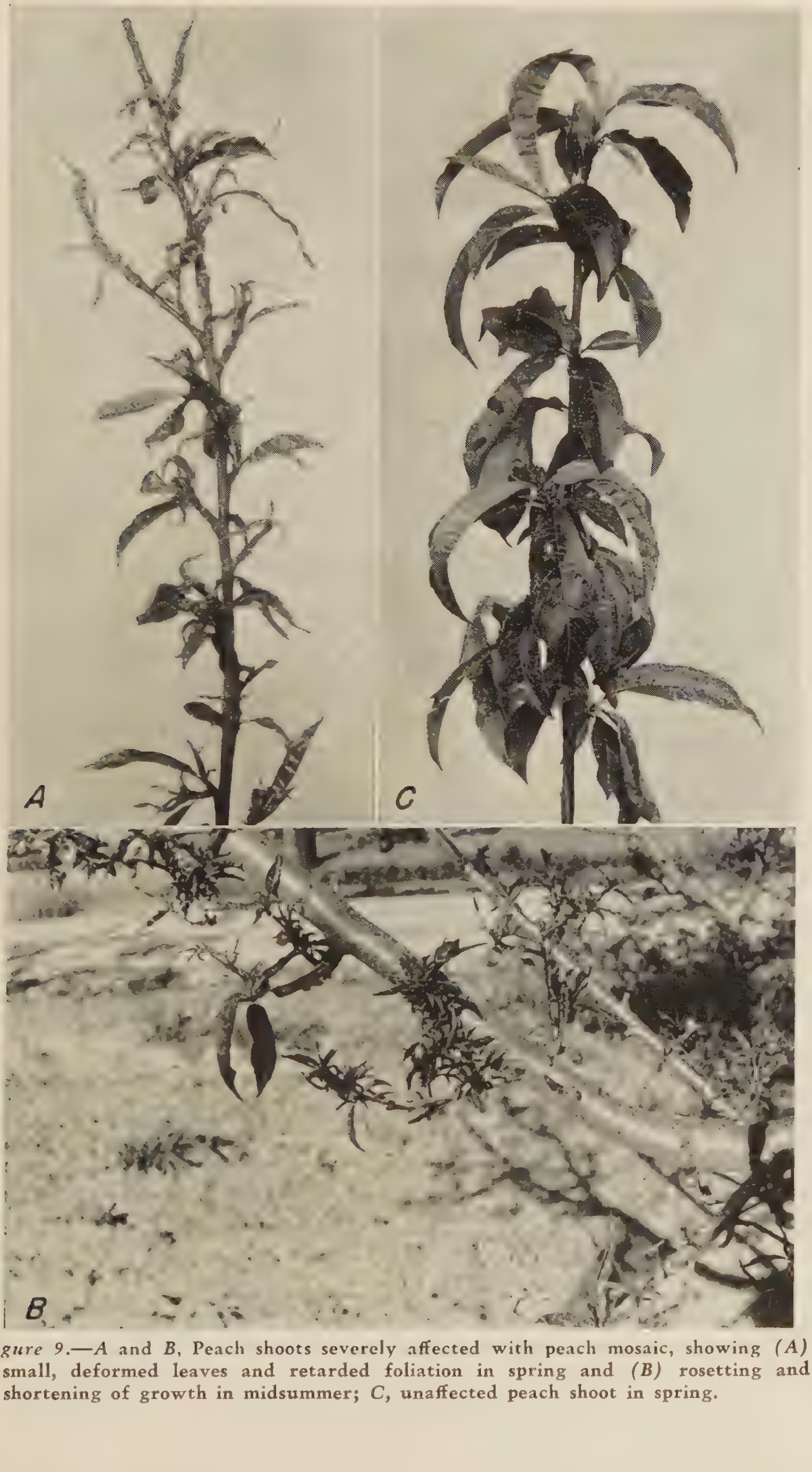

Figure 9.—*A* and *B*, Peach shoots severely affected with peach mosaic, showing *(A)* small, deformed leaves and retarded foliation in spring and *(B)* rosetting and shortening of growth in midsummer; *C*, unaffected peach shoot in spring.

retardation corresponds to the virus form involved (pl. 4, *D*). On severely affected peach trees, early-formed leaves may wither and fall or may assume irregular shapes and be variously mottled (fig. 8). On some trees the initial symptoms are small yellow flecks adjacent to veinlets (veinlet clearing); necrosis and dropping of tissue or whole leaves may be evident later. Usually

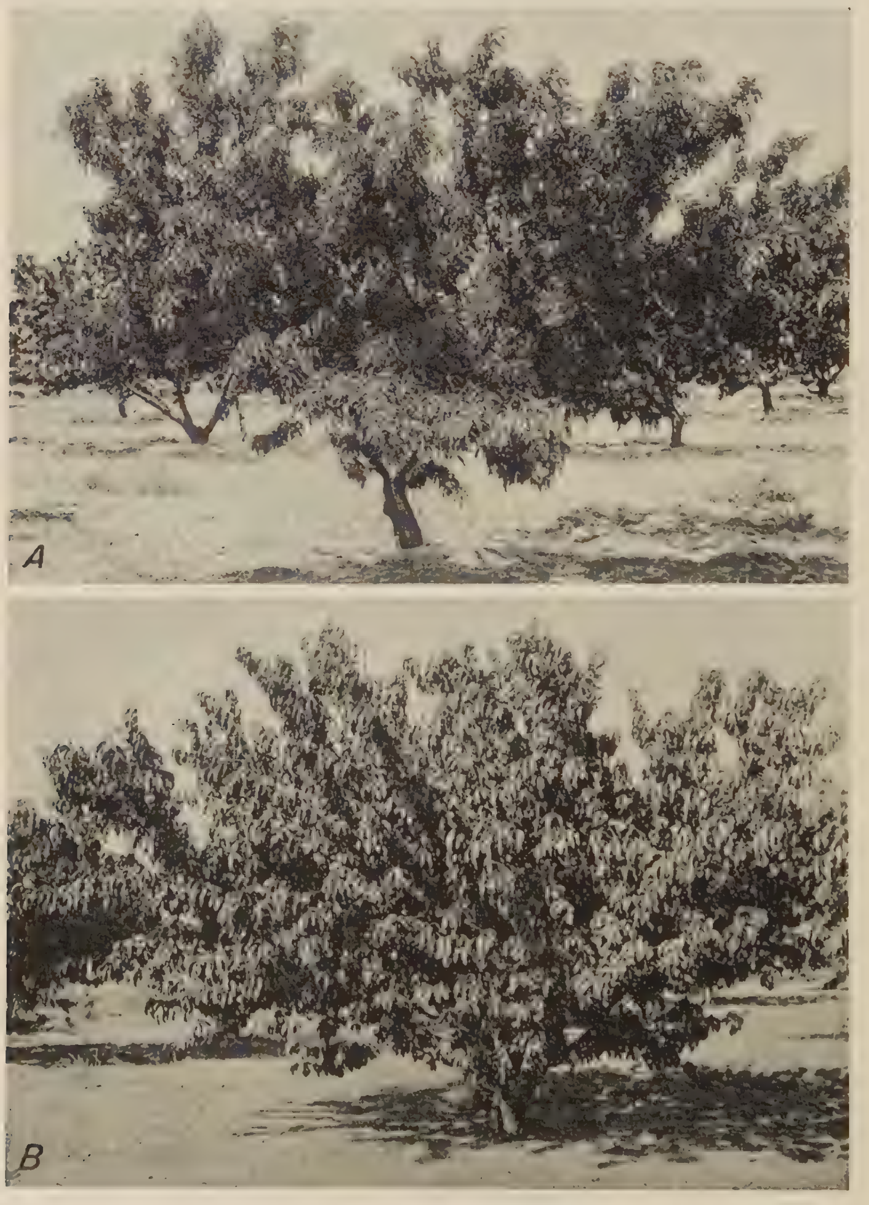

Figure 10.—*A*, Orchard Elberta peach tree affected with peach mosaic, in summer; *B*, comparable unaffected tree.

succeeding leaves on such trees are variously and strikingly mottled by irregular patterns of yellow and yellow green (pl. 4, *C*). Mottling of the severe

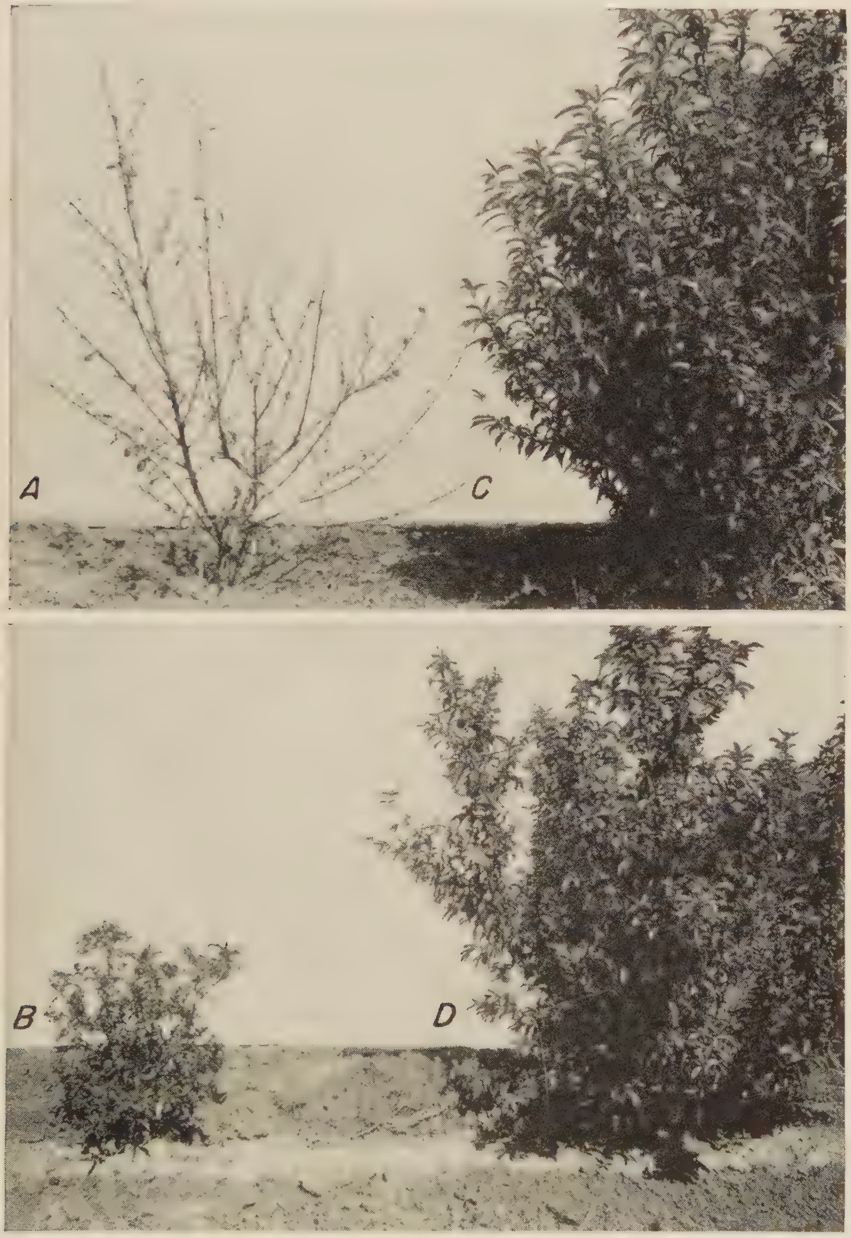

Figure 11.—J. H. Hale peach trees artificially infected with peach mosaic virus: *A*, With retarded foliation the first spring after inoculation; *B*, dwarfed, but without retarded foliation, the second spring after inoculation. *C* and *D*, Comparable unaffected trees.

type is usually accompanied by distortion and reduction in size. On mildly affected trees both mottling and distortion of leaves are less pronounced. The degree of mottling, presence of veinlet clearing, and amount of leaf distortion, retardation, and dwarfing appear to vary with different forms of the virus; thus the total effect on the tree varies. Some forms produce brilliantly mottled patterns, but fail to dwarf trees as much as others which produce less mottle. With some forms only scattered leaves may be affected, but these may be distorted and brilliantly mottled, whereas with others the majority of the leaves may be affected, but only mildly mottled and distorted.

As the season progresses, trees in the first year of the disease tend to become greener in contrast with normal than they were early in the season (*46, 114*). The probable reasons are that growing conditions are more favorable for the host and possibly conditions are less favorable for the virus. Succeeding shoot growth is less than normal (fig. 9), and affected trees become variably dwarfed (fig. 10), dwarfing corresponding to the severity of spring symptoms. By the second year, diseased trees show less shock in the spring and appear to go into a chronic stage of the disease (fig. 11). In this condition trees have more leaves than during the acute stages of infection, yet they show well-developed mottle, reduced shoot growth, and other symptoms corresponding to the form of the virus with which they are affected. The veinlet-clearing symptom occurs only on leaves on newly infected shoot growth and does not recur in growth diseased more than 1 year.

On varieties severely affected the fruits are usually small, bumpy, and misshapen (pl. 4, *B*). The bumps usually develop coincidentally with the stone-hardening stage. On less damaged varieties the fruits are smoother. Virus forms producing only mild effects, even on varieties like J. H. Hale, do not make the fruits bumpy.

Another striking effect of the peach mosaic virus is the production of white spots and streaks in the pink color in flowers of varieties having large, showy pink flowers (pl. 4, *A*). These spots are boat-shaped, and the larger have pink centers. The overlapping and variation in size of spots produce the appearance of variously shaped streaks and splashes. Such color patterns have not been seen on varieties having small or intermediate-sized flowers.

The amount of damage caused by peach mosaic to peach depends largely upon two factors—the form of the virus involved and the tolerance of the peach variety. Peach varieties may be divided into three comparative classes according to the degree they are damaged by forms which produce severe effects on the J. H. Hale variety. The most important commercial varieties are classified in table 1. Virus forms which produce only mild symptoms on J. H. Hale may exist without symptoms in less damaged peach varieties.

Other hosts.—Symptom expression on apricot, almond, myrobalan plum, Japanese apricot, American plum, chickasaw plum, Bessey cherry, Manchu cherry, and certain varieties of prunes and Japanese-type plums is sufficiently uniform for them to be treated as a group. On these hosts some forms of the peach mosaic virus make brilliant symptom patterns of rings and oak-leaf, splotchy, and irregular-shaped mottles. Others produce patterns similar to these in outline but less brilliant in intensity, while still others produce infection without any symptoms. Given enough sources of the virus, a symptom gradient for these species can be formed, ranging from no symptoms to brilliant, similar to that occurring on peach but without specific correlation with the effects on peach; that is, forms producing striking mottle on apricot may

TABLE 1.—*Classification of peach varieties according to the relative severity of their reaction to peach mosaic*

Severely damaged varieties	Medium-damaged varieties	Slightly damaged varieties
Afterglow	Admiral Dewey	Andora[1]
Ambergem	Alexander	Australian Saucer
Belle	Babcock	Carolyn[1]
Bonita	Brackett	Cortez[1]
Dixigem	Carman	Curry
Dixired	Champion	Erly-Red-Fre
Duke Hale	Cumberland	Fisher
Early Halehaven	Early Elberta	Gaume[1]
Elberta	Early Rose	Greensboro
Fay Elberta	Early Wheeler	Halford No. 2[1]
Fireglow	Eclipse	Johnson[1]
Gage Elberta	Fair Beauty	Lukens
Golden State	Fortuna[1]	Mikado
Goldeneast	Fredberta	Orange Cling[1]
Goldenglobe	Golden Jubilee	Paloro[1]
Halberta	Halehaven	Peak[1]
Hiley	Herb Hale	Phillips[1]
Illinois	Jerseyland	Shasta[1]
J. H. Hale	Krummel	Sims[1]
Kalhaven	Lizzie	Stanford[1]
Late Elberta	Lucas Beauty	Stuart[1]
Loring	Newday	Sullivan No. 1[1]
Meadow Lark	Ozark	Sullivan No. 2[1]
Nectar	Redelberta	Sullivan No. 4[1]
New Jersey State Rose	Redskin	Sungold
Pacemaker	Rochester	Tudor[1]
Prairie Rambler	Southland	Tuskena (Tuscan)[1]
Raritan Rose		Valiant
Redhaven		Walton[1]
Rio Oso Gem		Williams[1]
Roberta		Zuni
Shippers Late Red		
Sunday Elberta		
Sunhigh		
Vedette		
Veteran		
White Hale		
Wilma		

[1] Clingstone.

produce severe or only mild symptoms on peach and conversely. In general, the degree of symptom production on apricot (fig. 12, *A*) and on almond (fig. 12, *B*) by the various virus forms corresponds to the behavior of these forms on the other hosts in this group. Exceptions are certain varieties of cultivated plums and prunes, some of which develop symptoms with the apricot-symptom-producing forms, but not with those forms which fail to mottle apricot. Some plum varieties can be infected and retain any of the virus forms without symptoms. Some plum varieties appear immune. Plum and prune varieties that develop symptoms are Becky Smith, Beauty, Shiro, Flaming Delicious, Cain Seedling, French (Agen) (fig. 13), and Standard.

Transmission and Incubation Period

Transmission of the peach mosaic virus has been effected experimentally from peach to peach by tissue grafting with the following tissues: Fruit,

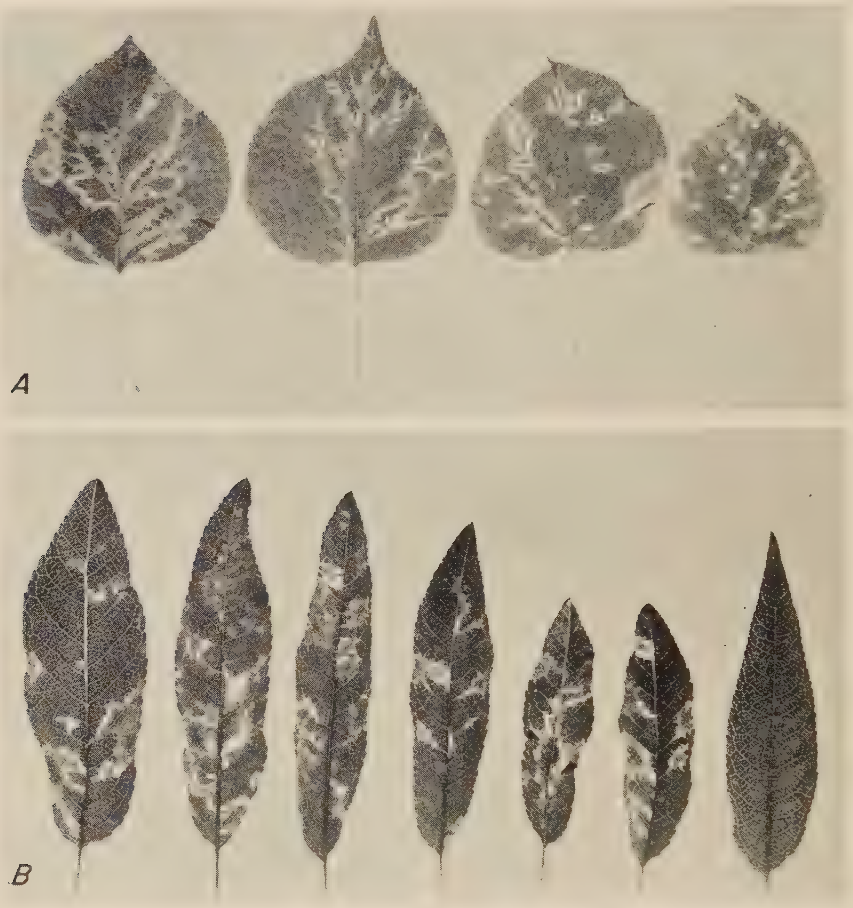

Figure 12.—**Leaves of *(A)* Royal apricot and of *(B)* Ne Plus Ultra almond affected with peach mosaic. Unaffected Ne Plus Ultra almond leaf at lower right.**

leaf, stem wood, stem bark, root wood, and root bark (*63*). It thus appears that nearly all the tissues of a peach tree are invaded. If scions of diseased tissue are grafted on healthy trees at the time of breaking of dormancy, transmission may result in as short a period as 14 days. If grafts are made when the leaves are partially expanded, the incubation period may be as long as 6 weeks; and if they are made as late as June, symptoms do not usually appear until the following season. Kunkel (*133*) showed that a minimum contact of 2 days between diseased and healthy tissues made at the cambium is needed for transmission to take place.

Spread in nature has varied with locality and is assumed to be through an insect vector. Little is known of the incubation period after infection under natural conditions. Bodine (*33*) reported a case in which seedlings planted in the spring showed symptoms in August. He assumed that they were probably infected in April or May.

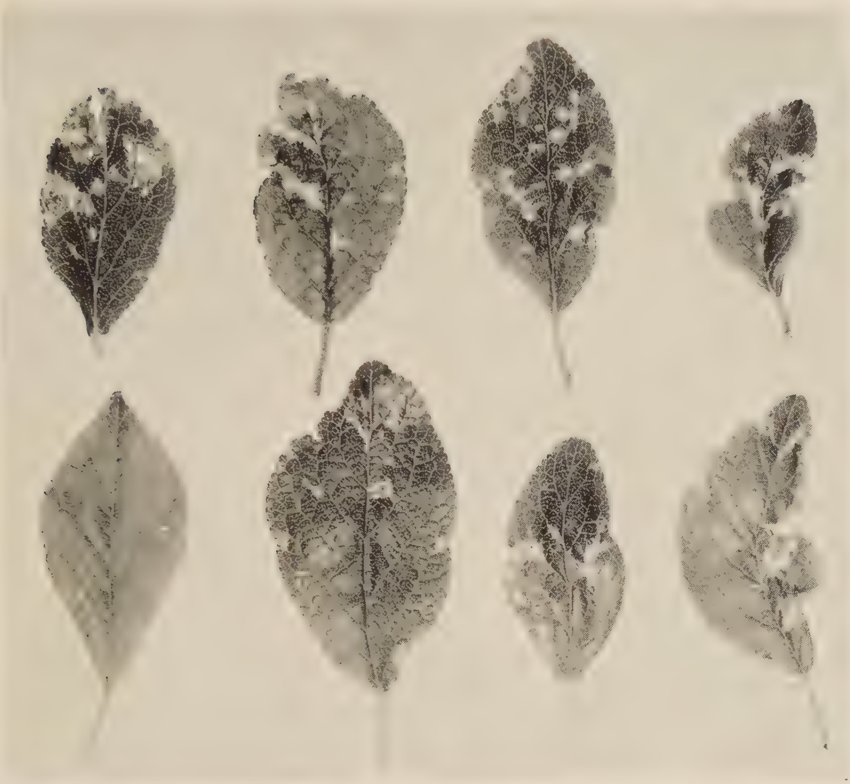

Figure 13.—Leaves of French (Agen) prune affected with peach mosaic. Unaffected leaf at lower left.

Thermal Relationships

Heat treatments of infected peach trees and bud sticks were not effective for the cure of peach mosaic, whereas similar treatments inactivated the viruses of peach yellows, peach rosette, little peach, and red suture (*132*).

Control Measures

The control of peach mosaic was first initiated as an eradication program. Later, survey results showed the disease to be widely existent, especially in noncommercial and marginal sections. In some districts the combined cost of inspection, removal, and loss from removal of trees of slightly damaged varieties overbalanced the economy of protection of newly planted orchards. In these districts growers are limited in the choice of varieties to those which tolerate mosaic with little damage. In some districts where growers desire Elberta and other nontolerant varieties, spread is being effectively held down by removal of infected trees. Removal is coordinated with a nursery-inspection and quarantine procedure for prevention of further spread through nursery stock within the infected districts and to sections outside.

Remarks

The peach mosaic virus comprises a group of many extremely variable forms which produce a wide range of symptoms that are very hard to delimit (*32*, *57*, *64*). New forms, the interaction of which appears to depend on their relationship, seem to be arising continually. Symptom expression is further complicated by the varietal reactions of the hosts. A given form of the virus will produce symptoms ranging from mild on one host to severe on another. Conversely, on a given host there will be a range of symptoms because one form of the virus produces mild symptoms and another severe. A person without experience might confuse with peach mosaic certain other virus diseases of the mosaic group, especially those caused by some forms of ring spot virus during the spring season, peach mottle, and yellow bud mosaic.

X-DISEASE [12]

By E. M. Stoddard, E. M. Hildebrand, D. H. Palmiter, and K. G. Parker

Other Common Names of the Disease

The disease has also been called eastern X-disease, yellow-red disease, yellow-red virosis, and eastern yellow-red virosis.

Names of the Causal Virus

Marmor lacerans Holmes; *Carpophthora lacerans* (Holmes) McKinney.

History and Geographic Distribution

X-disease was first reported on peach in Connecticut in 1933 (*216*), but reports from growers indicate that it had been present several years previously. The status of the disease in Connecticut from 1933 to 1939 was discussed by Stoddard (*216, 217, 218, 219, 220, 221, 222*). The disease was identified in Massachusetts and New York in 1938 (*41, 99*), in Ontario, Canada, Michigan, and Pennsylvania in 1941 (*6, 48, 158*), and in Ohio in 1944 (*78*).

X-disease has been reported on chokecherry in Connecticut (*222*), Illinois (*1, 203*), Indiana, Maine, Massachusetts (*42*), Michigan, New Hampshire, New York (*100*), Ohio, Pennsylvania (*268*), Rhode Island, Vermont, and Wisconsin (*222*) and in Ontario.

In New York growers reported that the disease was present on cultivated sour cherries as early as 1938. The disease was observed by Palmiter in 1942 and detailed inoculation studies were begun in 1946. Transmission to sour cherry was reported by Palmiter and Parker (*171*). X-disease has now been found in at least 36 cherry orchards located in 7 counties, but no definite survey has been conducted. It has been found in 1 Michigan cherry orchard, but its full extent on cherry in that State has not been determined.

On cultivated sweet cherry the disease has been observed with certainty on the Windsor variety in eight orchards distributed in three counties in New York and on the Bing variety in one orchard. It was considered severe in three of these orchards. Bud inoculations made from the Windsor variety to peach seedlings resulted in the development of characteristic symptoms of X-disease.

Economic Importance

X-disease is a major disease of peach in Connecticut, Massachusetts, and New York. It is present and potentially important in Michigan and Pennsylvania, but it is not considered of importance in Ontario, where few chokecherries are growing adjacent to peach plantings. Affected peach trees of bearing age become commercially worthless in 2 to 4 years. The younger the tree the more severe and rapid the development of the disease; consequently, trees infected at 3 years of age or less rarely produce fruit. Infected cherry trees soon become worthless for fruit production. In certain New York orchards up to 50 percent of the English Morello trees are not bearing as a result of this disease, and the disease has made many Montmorency trees unprofitable in other orchards.

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[12] The description of symptoms and other characteristics applies to X-disease as it occurs in eastern United States.

Host Range

Symptoms are produced on all observed varieties of peach (*Prunus persica*) (*102*); on purple-leaved peach (*P. persica* var. *atropurpurea*); on nectarine (*P. persica* var. *nectarina*); on chokecherry (*P. virginiana*); on almond (*P. amygdalus*); on wildgoose plum (*P. munsoniana*); on Chinese bushcherry (*P. japonica*); on Bessey cherry (*P. besseyi*) (*223*); on cultivated sour cherry (*P. cerasus*) varieties Montmorency, English Morello, and Chase; on mazzard (*P. avium*) seedlings; and on sweet cherry (*P. avium*) varieties Windsor and Bing. Other *Prunus* spp. are suspected of being hosts, but studies have not been completed. Kunkel (*136*) demonstrated transmission of the virus to and the production of symptoms on various herbaceous plants: tomato (*Lycopersicon esculentum*), carrot (*Daucus carota*), parsley (*Petroselinum crispum*), and periwinkle (*Vinca rosea*).

Wild American plum (*P. americana*) has been infected, but developed no visible symptoms. Wild black cherry (*P. serotina*) and beach plum (*P. maritima*) did not retain the virus after inoculation and were thus determined to be immune.

Symptoms

Peach.—Foliage of affected peach trees appears normal in the spring for about 7 weeks after growth starts; then suddenly a diffused and blended yellow and red discoloration, frequently accompanied by a longitudinal upward rolling of the leaf edges, appears. The discolored spots may occur anywhere on the leaf blades, are irregular in shape and size, and vary in number from few to many. At first the spots appear water-soaked, but soon they exhibit the characteristic coloration of blended red and yellow. Simultaneously the remainder of the leaf blade becomes chlorotic. The leaves become brittle; and the discolored areas usually fall out without necrosis, giving a characteristic tattered appearance to the foliage. Eventually affected leaves except the tip ones drop; the tip ones are retained to the end of the growing season (pl. 5). On greenhouse-grown peach trees leaf rolling, chlorosis, and stunting of growth are very marked but spotting, tattering, and defoliation are less than on trees grown out of doors. This is also true of seedling trees grown from so-called wild pits in the greenhouse or out of doors. Purple-leaved peach shows chlorosis and bleaching of the purple color and finally exhibits a blotchy yellow-green and purple coloration.

The fruits on affected parts of trees usually shrivel and drop soon after the foliage symptoms appear. The occasional fruits remaining on the trees ripen prematurely and are unpalatable in flavor, and seeds do not develop within the pits. Fruits on healthy growth of infected trees are unaffected.

Trees of bearing age are usually not completely infected at one time; rather, the affected twigs and branches are scattered indiscriminately over the tree, standing out in sharp contrast with the healthy portions of the tree. Viewed from a distance, infected trees have a distinctly lighter color than adjacent healthy trees. The infected, defoliated branches are undoubtedly weakened and consequently suffer from winter injury, resulting in a certain amount of dieback. Young trees usually die after infection, but old trees may live indefinitely.

Chokecherry.—In the first year of infection the foliage of chokecherry (pl. 6, *E*) shows dull to brilliant yellow, orange, and red coloration of the foliage by mid-June—6 or 7 weeks after growth starts—intensifying with the season and reaching its height in August. The brilliant foliage colors in the prevailing green background of the countryside in summer can be seen for considerable distances. In the second season of infection the symptoms are different. The leaves have a duller color, and stunted growth or rosettes appear on some of the terminals; such growth has leaves much smaller than normal, abnormal in shape, and often very rugose at the shoot tips. By the third season the most conspicuous symptoms are the duller colors and the terminal rosettes, on which dark green prevails. Death follows this advanced stage.

Diseased chokecherries may blossom; but most, if not all, of the ovaries abort at an early stage. The fruits which are retained at the end of the season do not mature and have undeveloped flesh and always dead seeds in the pits. When grown in the greenhouse, infected chokecherries show the advanced rosette stage of disease that frequently results in death during the second growing season.

Sour cherry.—In New York (*171, 172, 173*) X-disease has been transmitted from cherry to cherry, from cherry to peach, and from chokecherry to cherry. The disease is characterized by many different symptoms apparently resulting from a complication of factors, including varietal differences, the influence of rootstock, soil conditions, and seasonal temperature variations. The possible complex association of other viruses is also considered. Generally speaking, the disease is difficult to diagnose by foliage symptoms alone. The rootstock on which a variety is propagated plays an important part in symptom expression.

The symptoms on Montmorency on mazzard rootstock show first on a single branch which bears cherries of smaller size than normal, of lighter color, and frequently pointed (pl. 6, *A*, *B*). Some normal fruits are usually interspersed on portions of the same branch, especially during the first year.

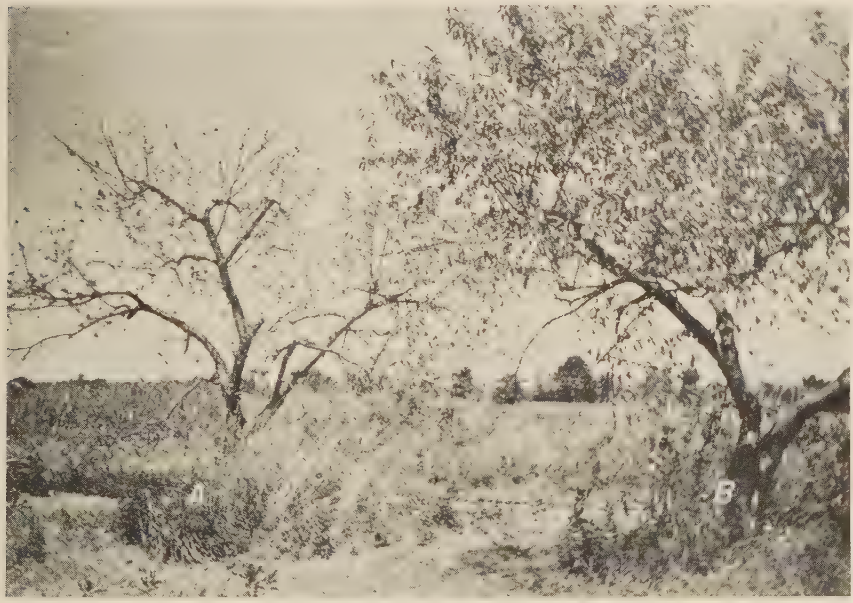

***Figure 14.*—Montmorency cherry trees affected with X-disease: *A*, Tree that had wilted 3 to 4 weeks before being photographed; *B*, tree just beginning to wilt.**

The disease increases progressively each year throughout an individual tree until finally most of the fruits fail to reach maturity. There is also a tendency toward rosetting of the shoots and a generally pale foliage.

The same uneven ripening of fruits of Montmorency on mahaleb rootstock is apparent, but less noticeable. In midseason the symptoms may include wilting of foliage, and later in the summer on some trees they may include decline in vigor (fig. 14). Young trees on mahaleb rootstock respond to inoculation with a rather rapid wilt and decline in the first season after inoculation. Mature trees on mahaleb rootstock which wilted in midsummer and showed dead leaves by fall were found to have a large proportion of dead roots. Root death proceeded progressively from the distal end toward the base, with dead bark overlying live wood. In contrast, roots dying from winterkilling or wet feet showed root injury initiated largely from the proximal end and progressing toward the tips, with wood discolored under live bark.

The symptom pattern of immature fruits of English Morello on mazzard rootstock follows that of Montmorency. However, the fruits are less pointed and more spindle-shaped. The fruits on diseased trees have a tendency to drop early. After a few years few or none remain on the diseased trees at harvesttime. Diseased trees of English Morello growing on mahaleb rootstock show wilt and decline and die rather quickly without the other symptoms developing to any marked degree.

Young English Morello on mahaleb rootstock when infected artifically wilts and declines as does Montmorency. When it was on mazzard, in one experiment a rosette type of growth (fig. 15), as observed on trees in the orchard, developed in the second year.

Sweet cherry.—So far, observations have been made on infected cultivated sweet cherry during only 1 year and in a relatively small number of orchards.

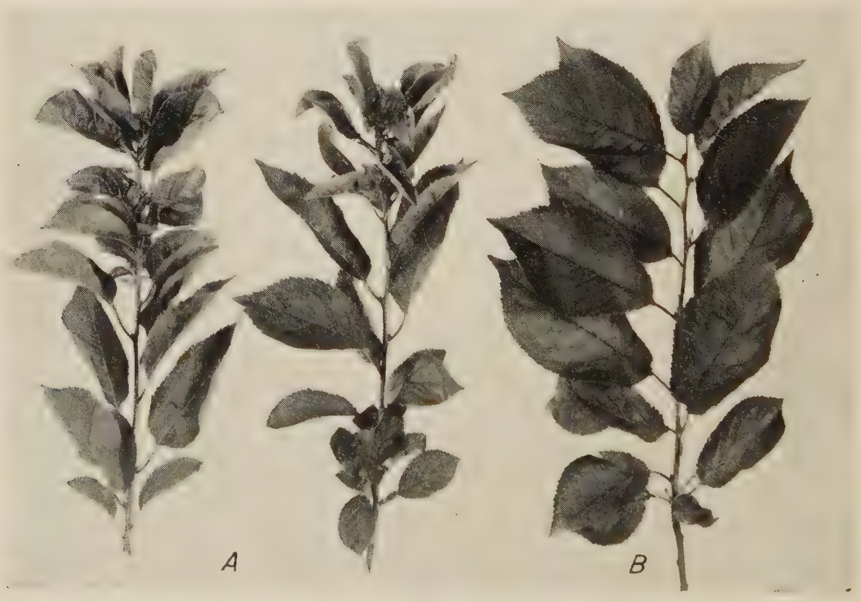

Figure 15.—*A*, Shoots from English Morello cherry tree affected with X-disease, showing rosette type of growth; *B*, comparable unaffected shoot.

WESTERN X-DISEASE

By E. L. Reeves, Earle C. Blodgett, T. B. Lott, J. A. Milbrath, B. L. Richards, and S. M. Zeller

Names of the Disease

Several common names have been applied to the disease caused by the western X-disease virus on different hosts. "Western X-disease" was applied originally to the disease as expressed on peach (*188*), and it has been used in referring to the disease on western chokecherry. In Utah western X-decline also has been used for the disease on peach. Red-leaf and western X red-leaf (*201*) have been applied to the disease on western chokecherry.

Wilt and decline (*196, 199*), western X wilt and decline, and decline and dieback of sour cherry have been applied in Utah to the disease as expressed on sweet or sour cherry trees growing on mahaleb cherry rootstock.

The name "little cherry" has been commonly used in connection with various diseases of cherries in which reduced size of fruit is a feature. It was applied first to a disease of cherries occurring in the Kootenay Lake area of British Columbia, Canada (*73*). It has also been commonly employed in referring to a disease of cherries in Idaho, Oregon, Utah, and Washington (*28, 29, 118, 185, 201, 254, 265*). There are no data to establish whether there is a relationship between western X-disease and the little cherry disease that occurs in British Columbia (described on p. 126). In both Utah and Oregon experiments have shown that the disease of the little cherry type affecting both sweet and sour cherry and the western X-disease of peach and western chokecherry are caused by the same virus. In Utah little cherry fruit symptoms occur only on diseased cherry trees growing on mazzard rootstock, and the names "Utah little cherry" and "western X little cherry" have both been applied to the disease. Experiments have likewise shown that a disease of the little cherry type affecting Montmorency sour cherry and several varieties of sweet cherry in Washington is an expression of infection with the western X-disease virus. Interhost-transmission studies are still incomplete, but inoculum from diseased sweet or sour cherry placed onto healthy peach resulted in the development of western X-disease symptoms, whereas similar inoculum placed onto healthy sweet cherry resulted in the development of little cherry type symptoms. Tests on attempted transmission from diseased peach to healthy cherry are incomplete.

In order to avoid confusion with the disease of sweet cherry called little cherry in the Kootenay Lake area of British Columbia, the disease having symptoms of the little cherry type on sweet and sour cherries associated with western X-disease of peach in Idaho, Oregon, Utah, and Washington is referred to in this section as "western X little cherry," a name previously used in Utah.

Name of the Causal Virus

"Western X-disease virus" is the common name proposed. No other name is suggested for the virus, pending clarification of the possible relationship to other viruses.

History

Blodgett (*12, 13, 18*) reported first observing the disease on peach in Idaho

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in 1936; and although transmission had not been demonstrated, he indicated that the symptoms were typical of X-disease as it occurred in the East.

In 1935 and 1936 Richards (*192, pp. 3–4; 193, pp. 82–83*) recorded abnormal reddening of chokecherry leaves in northeastern Washington. Subsequently the disorder was identified as western X-disease. In 1937 Richards recognized the same kind of red-leaf symptom on chokecherry in Utah.

In 1941 Richards and Hutchins (*197*) reported artificial transmission of the disease from peach to peach as a result of graft inoculations performed in 1939 and 1940 in Utah and indicated that symptoms resembled in some, but not in all, respects the X-disease described first in Connecticut (*221*).

Zeller and Evans (*258*) reported transmission of the disease from peach to peach in 1941, although they had first recognized the disease in eastern Oregon in 1939.

Bodine and Durrell (*37*) and Bodine (*34*) reported finding the disease in Colorado in 1940 and transmission from peach to peach in 1941.

In 1940 Reeves and Hutchins (*187*) published observations made in peach orchards in Washington and other Western States. They attempted to distinguish the true symptoms of western X-disease from false symptoms attributable to disorders of known, suspected, or unknown causes. Six groups were listed; groups 1 and 2 were designations for the disease now known as western X-disease, and a similarity of expressed symptoms to those of X-disease in the East (*221*) and buckskin (*180*), or leaf-casting yellows (*234*), in California was indicated; group 3 designated an unidentified disease; and groups 4, 5, and 6 designated diseases apparently attributable to several understandable causes, but with symptoms that could easily be confused with those of western X-disease. In 1941 Zeller and Evans (*258*) described marginal leaf spot of peach as a symptomatic expression of a distinct virus or a strain of the X-disease virus and indicated that it was doubtless the disease described under group 3 by Reeves and Hutchins (*187*). Jones (*96, pp. 32–33*) contributed information on marginal leaf spot, designating it as the most important peach disease in Washington and indicating that it was the same as the group-3 disease described by Reeves and Hutchins (*187*). In 1942 Lindner and Reeves (*139*) published data showing that the group-3 disorder described by Reeves and Hutchins (*187*) was not a transmissible disease, but was due to arsenic toxicity.

Rawlins and Thomas (*180*) recognized two types of buckskin of sweet cherry, one in Green Valley and another in Napa Valley of California. When either of these types was transmitted from cherry to peach, symptoms similar to those of X-disease developed on peach. The peach disease was first referred to as leaf-casting yellows (*234*). The possible relationship of western X-disease to buckskin has been indicated (*187, 258*) and is further discussed under Remarks.

Wilt and decline of sweet and sour cherries on mahaleb rootstock described for Utah (*196*) in 1945 was shown during 1941 to 1946 to be a virus disease induced by the virus responsible for western X-disease on peach and western chokecherry.

Western X little cherry occurring on either sweet or sour cherry on mazzard rootstock in Utah was attributed to the western X-disease virus (*198, 201*). The term "little cherry" was employed because of the failure of the fruit to reach normal size and maturity rather than because of any known relationship to the originally described little cherry (*73*) in British Columbia. The name "little cherry" was also applied to the disease in Washington (*28, 29, 185,* *254*), Idaho (*118*), and Oregon (*263, 265*) without reference to the rootstock on which the trees were growing. In Oregon Zeller and Milbrath (*265*) indicated the possible relationship of western X-disease of Montmorency cherry which bore small, late-ripening fruits to buckskin of sweet cherry in California (*180*).

On the basis of records for 10 years Lott found that in British Columbia some peach trees became diseased and later showed weakness without leaf symptoms, while a few affected trees returned to an apparently normal condition. On some trees leaf symptoms reappeared after having failed to appear for a year or two. Lott (*142*) described small bitter cherry, a disease of sweet cherry occurring in orchards adjacent to western-X-diseased peach in British Columbia, but no definite transmission information has yet been obtained to connect small bitter cherry and western X-disease.

Zeller (*256*) indicated that western X-disease of peach in Oregon probably consisted of at least two forms. In Washington Reeves collected western X-disease virus from a number of peach trees in different districts and found considerable variation in the effects of the disease produced on comparable experimentally infected peach trees; this variation suggested the probability of different forms of the virus. Some of these forms may be closely related to or the same as the virus causing other described diseases of the western X-disease group. It is recognized that further investigations are necessary to clarify satisfactorily the relationship of western X-disease to other named virus diseases of stone fruits, particularly pink fruit of sour cherry in western Washington, little cherry in British Columbia, albino in southern Oregon, small bitter cherry in the Okanagan Valley of British Columbia, buckskin in California, and X-disease in the East.

Geographic Distribution

Western X-disease as referred to in this section has been reported in British Columbia, Washington, Oregon, Idaho, Utah, and Colorado (*34; 37; 96, pp. 29–31; 119; 154; 184; 194; 197; 258; 260*). Similarity of the symptoms described for buckskin to those of western X-disease on both peach and cherry (*180, 234, 265, 265a*) indicates that California should be included.

In British Columbia the disease is limited to the southern part of the Okanagan Valley; in Oregon and Washington it occurs east of the Cascade Mountains except on the affected cherry trees reported in one location near Mount Vernon, Wash. (*254*). The disease is more widely distributed on western chokecherry than it is on peach and cherries, because the natural range of this plant is wider than the planting ranges of peach and cherries. On peach, cherries, and chokecherry the disease occurs in both northern and southern Idaho; in Utah it occurs in the great Salt Lake Basin; and in Colorado it occurs principally in Grand County.

Economic Importance

Western X-disease is considered to be the most serious virus disease of peaches in British Columbia, Washington, Oregon, Idaho, and Utah.

A survey of Washington peach orchards, conducted in 1942 and reported by Coe (*66*), showed that 60 percent of the orchards inspected had western-X-disease-affected trees and that on an average 2.4 percent of the trees in those orchards were diseased.

In the Okanagan Valley of British Columbia individual-tree records showed that 2.7 percent of 2,456 peach trees were diseased in 1940. By 1949 an additional 6.9 percent of the trees originally surveyed had shown disease. In

1945 a survey showed that 1.5 percent of 35,469 peach trees were diseased and that in orchards adjacent to wild hillsides up to 12 percent were affected.

Western X-disease is a serious disease of peach in Baker, Malheur, Wasco, and Umatilla Counties of eastern Oregon and is particularly serious on sweet and sour cherries in Wasco County. Recent surveys showed that 25 percent infection in sweet and sour cherry orchards is not uncommon and several orchards have nearly every tree affected. The number of infected cherry trees is increasing at an alarming rate, and infection in many orchards is doubling each year.

A survey of Washington cherry orchards made in 1947 (*29*) showed 1,491 diseased trees located on 295 properties. In a similar survey made in 1948 (*254*) 1,933 diseased trees were found on 344 properties. The percentage of infection was reported highest in Asotin County and in the suburban districts of Wenatchee and Yakima (*254*).

In 1943, 90 percent of the peach orchards in Davis, Box Elder, and Weber Counties, of Utah, were found to have diseased trees and some orchards had up to 83 percent of the trees affected (*191*). Observations in these same counties during 1947 and later (*200a*) showed that sweet and sour cherries on mahaleb rootstock had a high incidence of disease, that many trees were dead, and that others were economically unprofitable and showed characteristic wilt and decline symptoms. Sweet and sour cherries on mazzard rootstock also were severely affected. A few older orchards of cherries on mazzard rootstock had up to 100 percent of the trees affected and, although they were seldom killed by the disease, the trees were generally unproductive, the vegetative growth was reduced, and the life of the tree was apparently shortened. The greatest incidence of diseased cherry trees in Utah coincided with the occurrence of the disease on both peach (*190*) and chokecherry.

In Idaho western X-disease is serious on peaches in all the principal peach-growing districts and several peach orchards have been removed because of the disease. The disease occurs on cherries in all of the principal cherry-growing districts, but generally the percentage of infection is low, and many orchards are not affected. In a few orchards the disease has affected up to 50 percent of the cherry trees.

Host Range

Western X-disease occurs on peach (*Prunus persica*), all varieties commonly grown in the Northwest and other varieties and seedlings tested; nectarine (*P. persica* var. *nectarina*); sweet cherry (*P. avium*), all varieties commonly grown in the Northwest and other varieties and seedlings tested; sour cherry (*P. cerasus*) varieties Montmorency, Early Richmond, and English Morello; and western chokecherry (*P. virginiana* var. *demissa*). In Utah the virus has been transmitted by grafting to the eastern chokecherry (*P. virginiana*), Bessey cherry (*P. besseyi*), and Manchu cherry (*P. tomentosa*).

Simonds (*204*) reported successful transmission to the Jones and Early Horne varieties of apricot (*P. armeniaca*) and to Big Mack (probably *P. insititia*) and Duarte-Satsuma hybrid (probably *P. munsoniana* × *P. salicina*) plums. Reeves and Hutchins (*188*) and Reeves (*184*) reported negative results in attempted transmission to Moorpark apricot and Italian Prune (*P. domestica*). In Utah[13] apricot growing on peach rootstock was inoculated with buds from diseased sweet cherry trees. No apparent symptoms developed on the apricot, but western X-disease symptoms appeared on shoots growing from the peach rootstock.

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[13] Wadley, B. N. Unpublished data.

Symptoms

Peach.—Specific leaf symptoms generally become apparent on affected trees from early June through July, depending somewhat on the season. In Washington Reeves found what appeared to be different forms of western X-disease virus on peach. Trees infected with the severe form exhibited foliage symptoms 2 to 3 weeks earlier than those infected with milder forms. Also trees infected with the severe form were often killed the second or third year after infection, whereas those infected with a milder form were seldom killed, but showed a slow decline over a period of years. Peach trees do not become uniformly affected during the first year, but some twigs or branches exhibit symptoms while others appear normal. Plate 7, *C*, shows a typical example of a Salwey peach tree with some branches severely affected and others healthy. The initial expression of the disease during the first year may be evident in only a single branch or shoot. Affected twigs or entire branches frequently die during the winter; such dying, together with growth during subsequent years, gives the tree a one-sided and scraggly appearance.

In plate 8, *B*, is shown an Elberta peach tree in an advanced stage of the disease. This is representative of a second- or third-year period of infection by a severe form of western X-disease virus or of five or more years after infection by a milder form.

In general, the initial symptoms in early summer and those occurring on leaves on rapidly growing shoots throughout the season consist of irregular pale-green areas of varying size and shape in the lamina of the leaves. These areas may involve large portions of the leaf blade, which may separate from the normal leaf tissue and fall out, producing a shot-hole or tattered condition of the affected leaves (pl. 7, *A*). Leaves so affected remain pale green and seldom develop the red and yellow colors characteristic of the disease later in the season. Affected leaves become detached readily, and early defoliation may result.

On the leaves developing symptoms later in the season, water-soaked areas appear in the lamina; these areas develop into necrotic spots of various sizes and shapes. These necrotic areas may or may not drop out. The necrotic tissue takes on a tan or dark-brown color, sometimes surrounded by a purplish border (pl. 9, *top*), especially in some varieties having a red fruit color. The remaining portion of the affected leaf blade turns a greenish-yellow color and has irregular spots, streaks, or splashes of red. In late season the reddish color frequently follows the veins. Leaf fall is far less than during the earlier expression of the disease, and retention of affected leaves gives the tree a characteristic yellowish to yellow-red appearance.

The margins of the leaves of most varieties often roll upward, but on Lovell seedling peach the leaves sometimes remain flat and clear yellow with little mottling or necrosis. The amount of defoliation varies; defoliation may be complete or nearly so on individual twigs and seriously affected branches. During the late growing season other branches not so severely affected may retain considerable foliage, but this foliage becomes prematurely yellow in the fall. In plate 7, *B*, a branch of Orange Cling peach with some leaves having already dropped, but with considerable yellow and orange-yellow foliage remaining, is shown.

Fruits on severely affected branches usually shrivel and drop shortly after the appearance of the first leaf symptoms. Fruits usually remain attached on less severely affected branches and on branches which do not develop symptoms until late in the season. Fruits on the less severely affected branches fail to attain full size, ripen early, and sometimes fall from the tree 10 days to 2 weeks prior to the time of normal harvest. Such fruits are undersized and somewhat conical in shape, have withered apexes (pl. 8, *A*), and are insipid and bitter in flavor. Seeds of affected fruits fail to develop, and pits are discolored.

Western chokecherry.—Symptoms on western chokecherry develop in late June and July and show first as a greenish-yellow coloration. Later in the season the leaves acquire a bright-red coloration. During the second year of infection the leaves have a duller color, being mostly yellow with a little red. Growth is stunted, and the leaves are smaller (pl. 8, *D*). Fruits, when they occur, are smaller and more pointed than normal and in general exhibit the little cherry type of symptom found on affected sweet and sour cherries (pl. 8, *C*). Affected chokecherries may die out rather rapidly in some districts or may persist in a declining condition for many years.

Sweet and sour cherries on mahaleb rootstock.[14]—In Utah western-X-disease-affected sweet and sour cherry trees growing on mahaleb rootstock may wilt at any time during the growing season. The disease may be detected as early as late April or early May by the light-green or yellow leaf color and by the flaccid appearance of the leaves (pl. 10, *C, D*). Wilting may be so rapid that, as shown by the middle tree of plate 10, *D*, the leaves die without forming an abscission layer, become reddish brown, and persist on the tree throughout the summer and following winter.

Frequently, affected trees wilt slowly and retain their leaves until later in the season. Such trees may continue to live in a weakened, or declined, condition for several years. Leaves on slowly declining trees are retarded in development, smaller in size, more linear in shape, lighter green in color, and more sparsely distributed on the tree than normal. Defoliation occurs much earlier in the fall on affected trees than on normal ones of the same variety.

Diseased trees in the early stage of decline have a tendency toward delayed but excessive blooming and a heavy fruit set (pl. 11, *E*). Such declining trees may wilt abruptly and die after this excessive fruit setting. Fruits color earlier than normal, but they seldom develop full normal color or size and appear to be more elongated. Roots of severely diseased trees are always damaged. Usually the small rootlets and the cortex of the root are dead or decayed by late fall and the xylem is darker in color than that of healthy roots. Affected roots die progressively from the tips to the trunk.

Sweet and sour cherries on mazzard rootstock.—In Utah the symptoms on sweet or sour cherry on mazzard rootstock affected with western X-disease are most evident on the fruits although the foliage and the growth of the trees are also affected. Affected fruits are seldom more than half normal size and are inclined to be more pointed than normal and retain the pinkish-red or yellowish-green color of immature fruits far beyond the normal picking date (pl. 11, *A*). Affected fruits often exhibit a dull luster rather than a normal glossy appearance. They also definitely lack the flavor and sweetness of normal cherries. Affected fruits remain attached to the tree long after normal fruits are harvested, but they never acquire normal color.

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[14] The information given on sweet and sour cherries on mahaleb rootstock is limited to observations in Utah.

In Utah (*201*) symptoms in addition to reduced size of fruits are often associated with the disease, particularly on sweet cherry. Stipules may become elongated and broadened and persist much longer than on healthy trees; bud scales may remain attached several weeks after the buds open in the spring; and internodes on new growth may become shortened and thus give the trees a rosetted appearance. Leaves may be somewhat smaller and paler green than healthy leaves of the same variety. A few terminal buds break dormancy in late fall, and thus secondary growth results.

In Washington (*29*) symptoms in addition to those exhibited by the fruits are recognized as associated with western X-disease even though positive diagnosis of affected trees depends upon fruit symptoms. These additional symptoms become apparent after harvest. They have been found on severely affected trees and are absent or nearly so on lightly affected trees. The most noticeable of these symptoms is a fading of the dark-green color of some leaves to a yellowish green, which later becomes light brown to bronze. The coloration of the foliage is variable; a few leaves may appear severely sunburned, others are light brown, and about half of the foliage appears nearly normal or dull green. The bronzing of the foliage on severely affected trees readily distinguishes them from trees with normal-appearing foliage. Other symptoms that have been observed are separation of the bud scales (particularly on spurs previously bearing small fruits), which results in such buds having a frayed appearance, late blossoming, and a variable percentage of the terminal buds breaking into secondary growth. In Washington an occasional stipule may be found slightly elongated and broadened but the shape change is much less pronounced than in Utah. Also in Washington affected trees are not found with a pronounced rosetted appearance as they are in Utah.

In plate 9, *bottom*, western-X-little-cherry-affected fruits from an affected Bing tree in Washington are shown in comparison with normal fruits taken from the same tree. The relative percentage of small fruits on affected trees varies with variety even when the entire tree is generally affected. Severely affected Lambert and Black Republican trees have been observed with no fruits of normal size, but it is not unusual to find a severely affected Bing tree with 20 to 30 percent of the fruits apparently normal or nearly so. In plate 11, *B* and *C*, western-X-little-cherry-affected fruits from affected Napoleon (Royal Ann) and Lambert trees in Washington are shown in comparison with normal fruits taken from the same trees. Fruits from a severely affected Lambert tree are often conical or triangular in shape.

The first year a sweet or sour cherry tree exhibits symptoms of the disease, it is not unusual to find the small cherries confined to one or two branches. In other instances, especially on small trees, nearly every fruit is affected the first year that symptoms appear.

In Oregon the symptoms on sweet cherry are much the same as described in Washington. There are no reliable foliage symptoms and no appreciable decline in sweet cherry trees for several years after infection. On the Montmorency variety of sour cherry there is a rapid decline followed by dieback, a typical example of which is shown in plate 11, *D*. An infected tree produces no marketable fruit the second or third year after infection. The right side of the tree shown has seriously declined and many small limbs are dead, while relatively good foliage remains on its left side. In plate 10, *B*, fruits from an affected Montmorency tree in Oregon are shown in comparison with normal fruits from the same tree.

Transmission

Transmission has been reported by grafting or some adaptation of the grafting process and by the geminate leafhopper (*Colladonus geminatus* (Van D.)) (*252a*).

On peach there is considerable variation in the percentage of positive infections in graft-transmission experiments, depending upon the season of inoculation, the type of inoculum employed, the length of time the inoculum has been held after removal from a diseased tree before transmission is attempted, and probably the vigor of the tree being inoculated. The percentage of positive infections obtained is relatively high with inoculum taken early in the season as soon as recognizable symptoms appear and relatively low or zero in late summer, early fall, and the dormant season.

In early work in Oregon, after an incubation period of 2 years, only 4 cases of western X-disease resulted from Zeller's inoculation of 32 peach seedlings with inoculum from naturally infected western chokecherry. Later Zeller and Milbrath (*265*) made successful interhost transmissions of several diseases occurring in Wasco County, Oreg., by grafting and decided that western X-disease of peach, red-leaf of western chokecherry, and "little cherry" of sour and sweet cherry occurring there are all caused by the same virus. They also found that peach trees conditioned by prior infection with latent viruses commonly occurring in cherry trees gave a higher percentage of western X-disease upon receiving inoculum from western-X-disease-affected sweet or sour cherry trees.

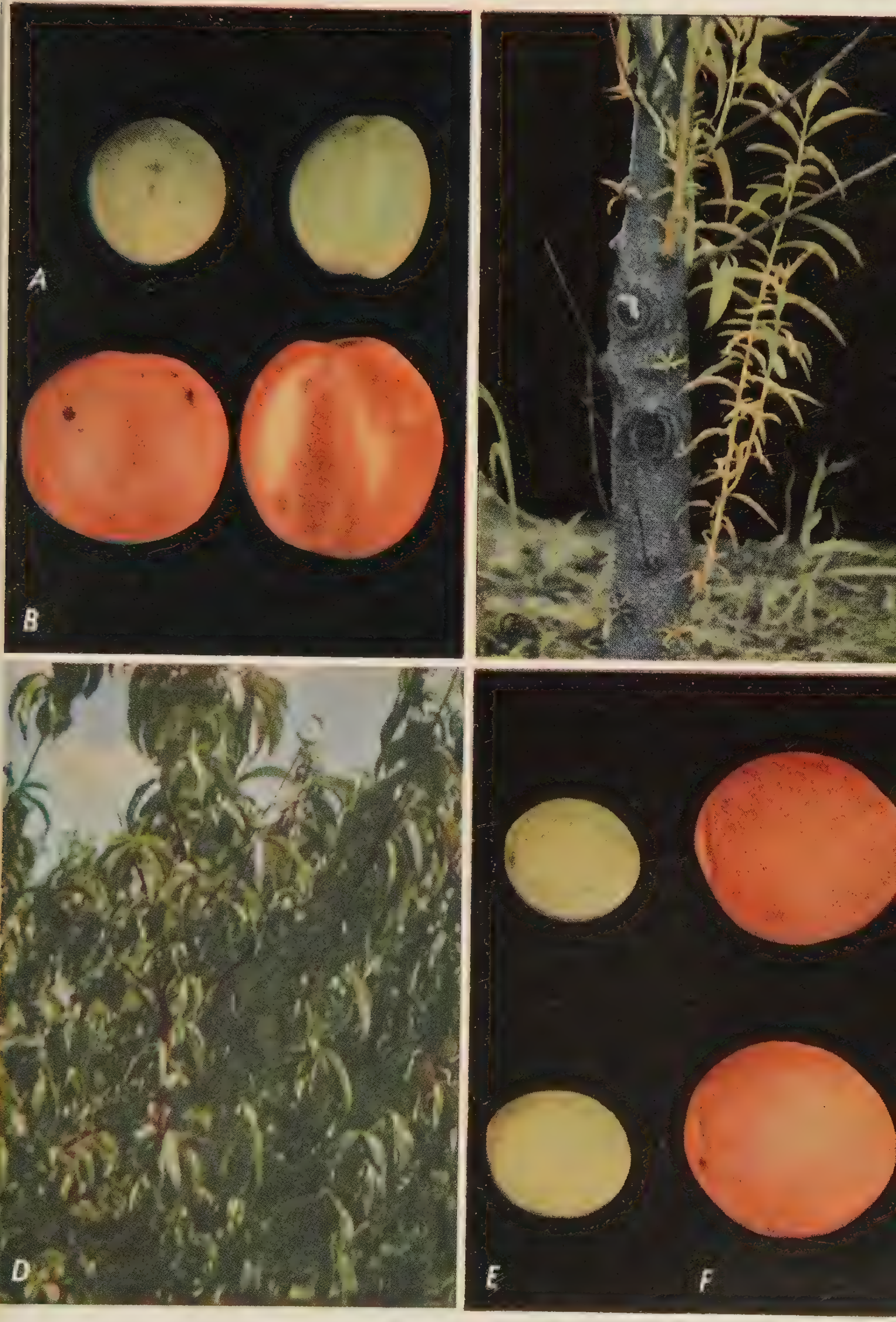
In Utah (*196, 199*) western X-disease virus from western chokecherry has been repeatedly inoculated into nursery trees of sweet and sour cherries growing on mahaleb rootstock and wilt and decline has resulted. Wilt and decline has likewise been induced on English Morello cherry by western X-disease virus taken directly from peach and indirectly on sweet and sour cherries from the peach after passage through western chokecherry.

By inoculation with buds and fruit spurs from diseased sweet and sour cherry trees in Utah (*198, 201*), typical western X-disease symptoms were produced on peach, typical red-leaf and characteristic small fruits on eastern and western chokecherries, and wilt and decline on sweet and sour cherries growing on mahaleb rootstock. The wilt and decline produced appeared identical with that induced by the western X-disease virus from western chokecherry and with that occurring in orchards.

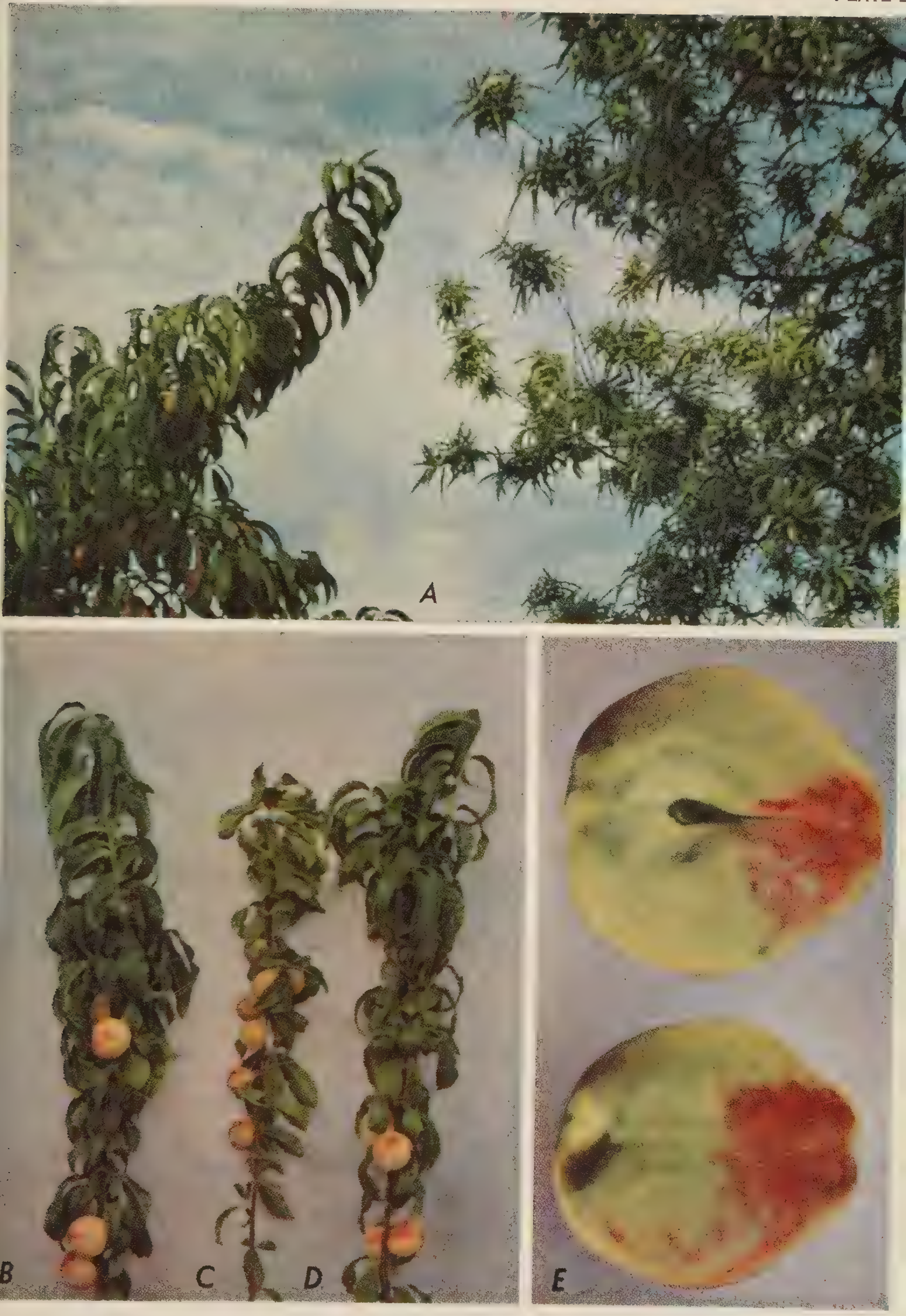
In Utah when normal sweet or sour cherry is top-worked on individual branches of a mahaleb tree and some of the resulting-variety branches are separately inoculated with western X-disease virus, the branches thus infected develop wilt and decline but the uninoculated branches remain normal. In plate 10, *A*, is shown a tree of mahaleb understock with three arms which had normal Bing buds inserted near a point indicated by the white tag just above the crotch on the branch at the right. The two outside branches, subsequently inoculated with buds taken from diseased western chokecherry show characteristic wilt and decline. The center branch, which was not inoculated, remained normal.

Incubation Period

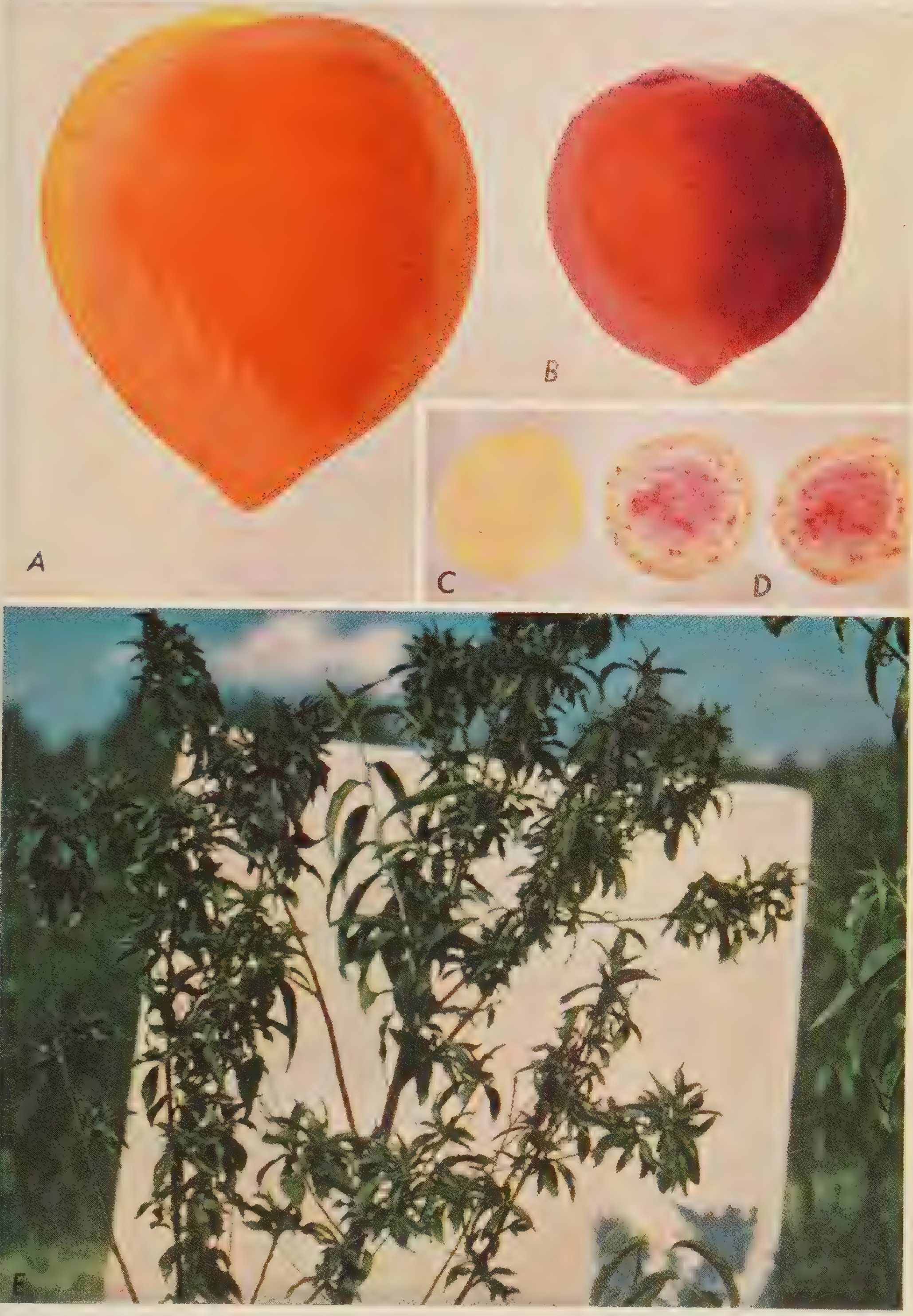
Peach trees inoculated with western X-disease virus in late summer by budding exhibited symptoms the following year at the usual time when symptoms of the disease appear. In 1941 Richards and Hutchins (*197*) reported as follows: "Of 40 Elberta nursery trees inoculated on July 26, 1940,



A, Normal immature peach fruits; B, comparable fruits affected with peach yellows, showing early maturity; C trunk of peach-yellows-affected peach tree, showing wiry shoots with small leaves; D, portion of little-peach affected peach tree, showing clustered, drooping leaves; E, immature fruits from Elberta peach tree affecte with little peach; F, comparable normal mature fruits.

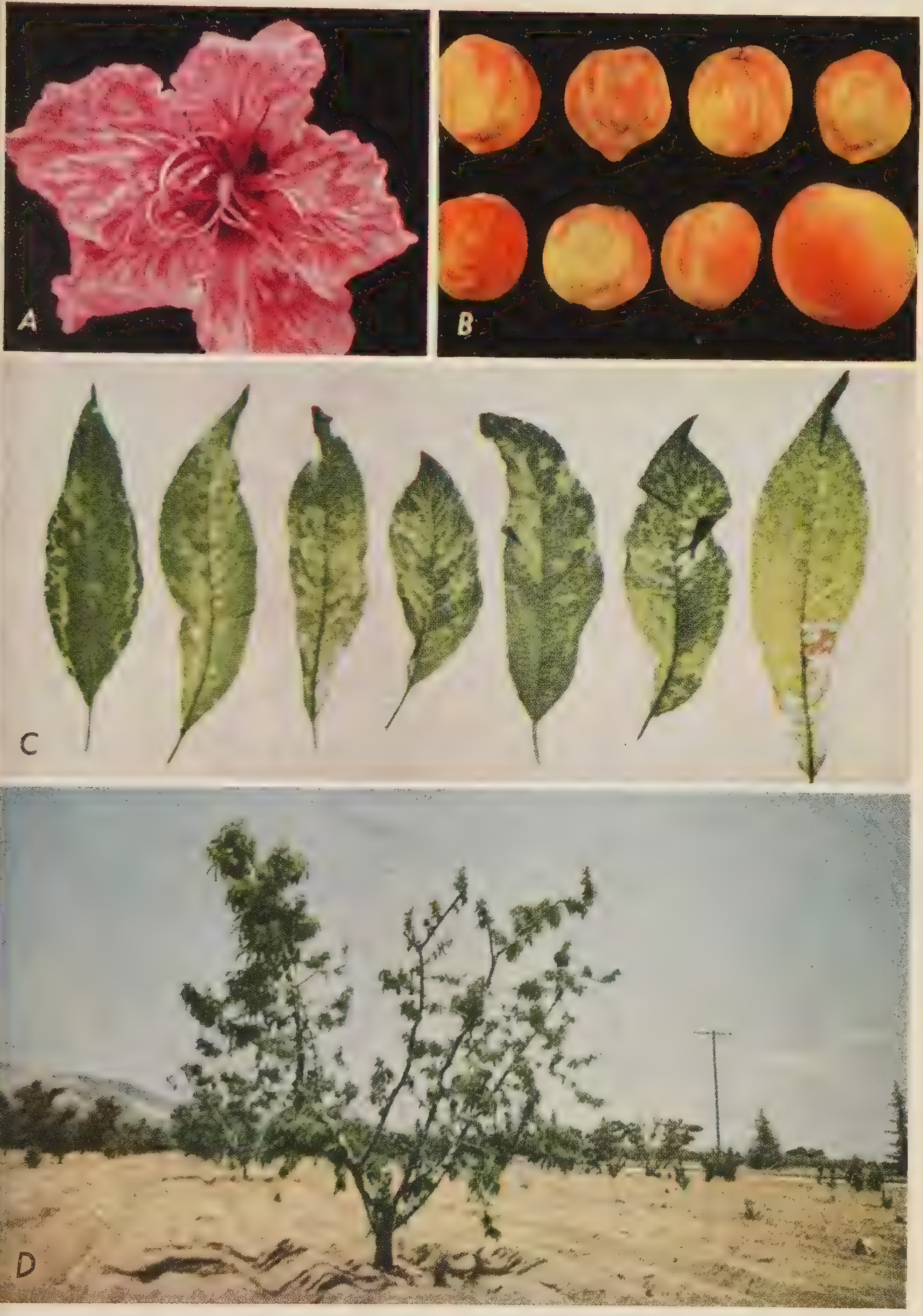


, Elberta peach foliage affected with peach rosette, in contrast with normal at left. *B–D*, Comparable peach shoots: *B*. From normal tree, with slightly immature fruits; *C*, from little-peach-affected tree, showing drooping foliage and immature fruits; *D*, from red-suture-affected tree, showing fruits colored on the suture side. *E*, Peach fruits showing enlarged, reddened suture side characteristic of red suture.

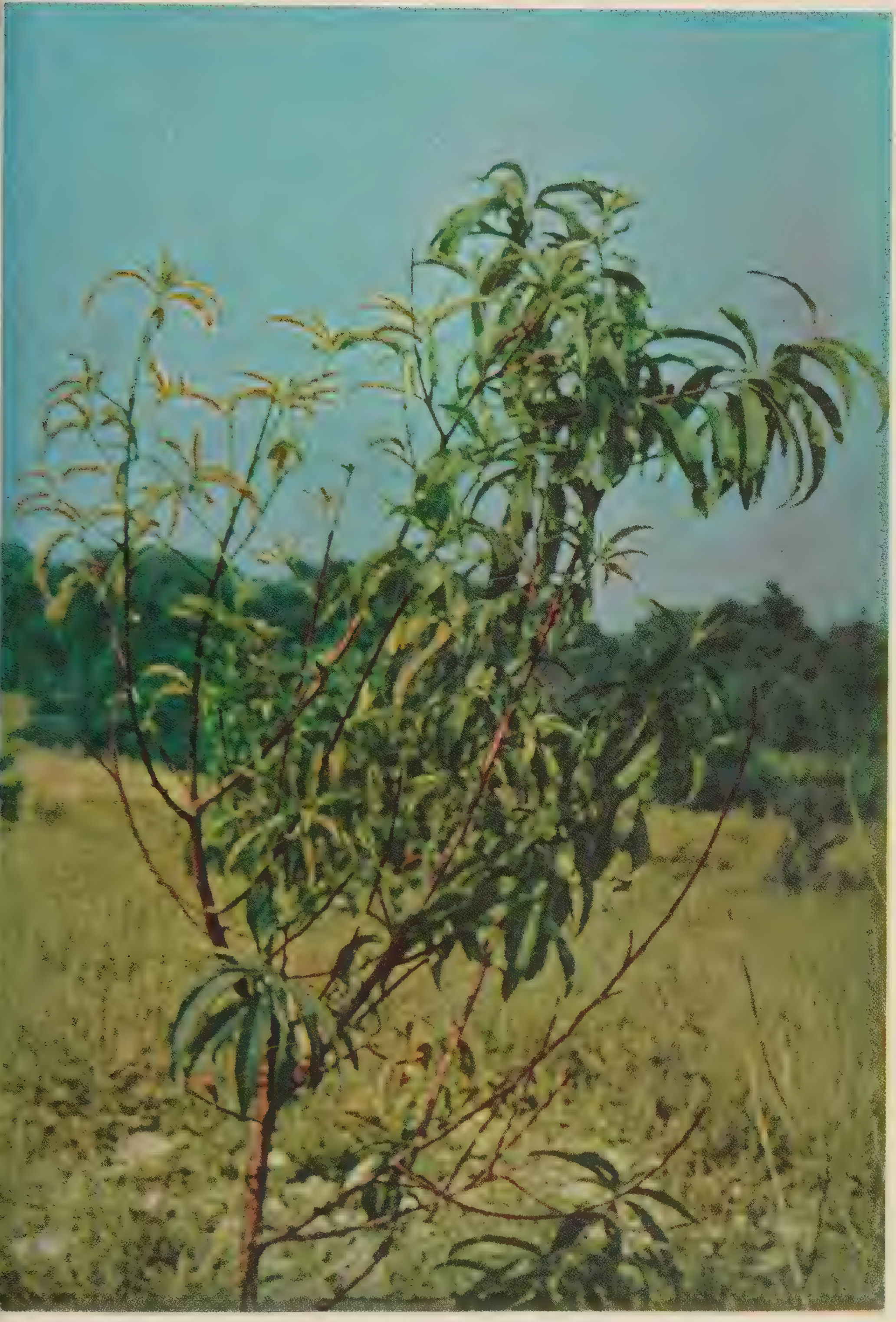


A, Normal Hiley peach. *B*, Comparable fruit from phony-affected tree. *C* and *D*, Transverse serial sections of root from phony-affected peach tree: *C*, Before chemical identification test; *D*, after test, showing color reaction in the woody cylinder. *E*, Three-year-old J. H. Hale peach tree affected with rosette mosaic, with a normal shoot in the center.

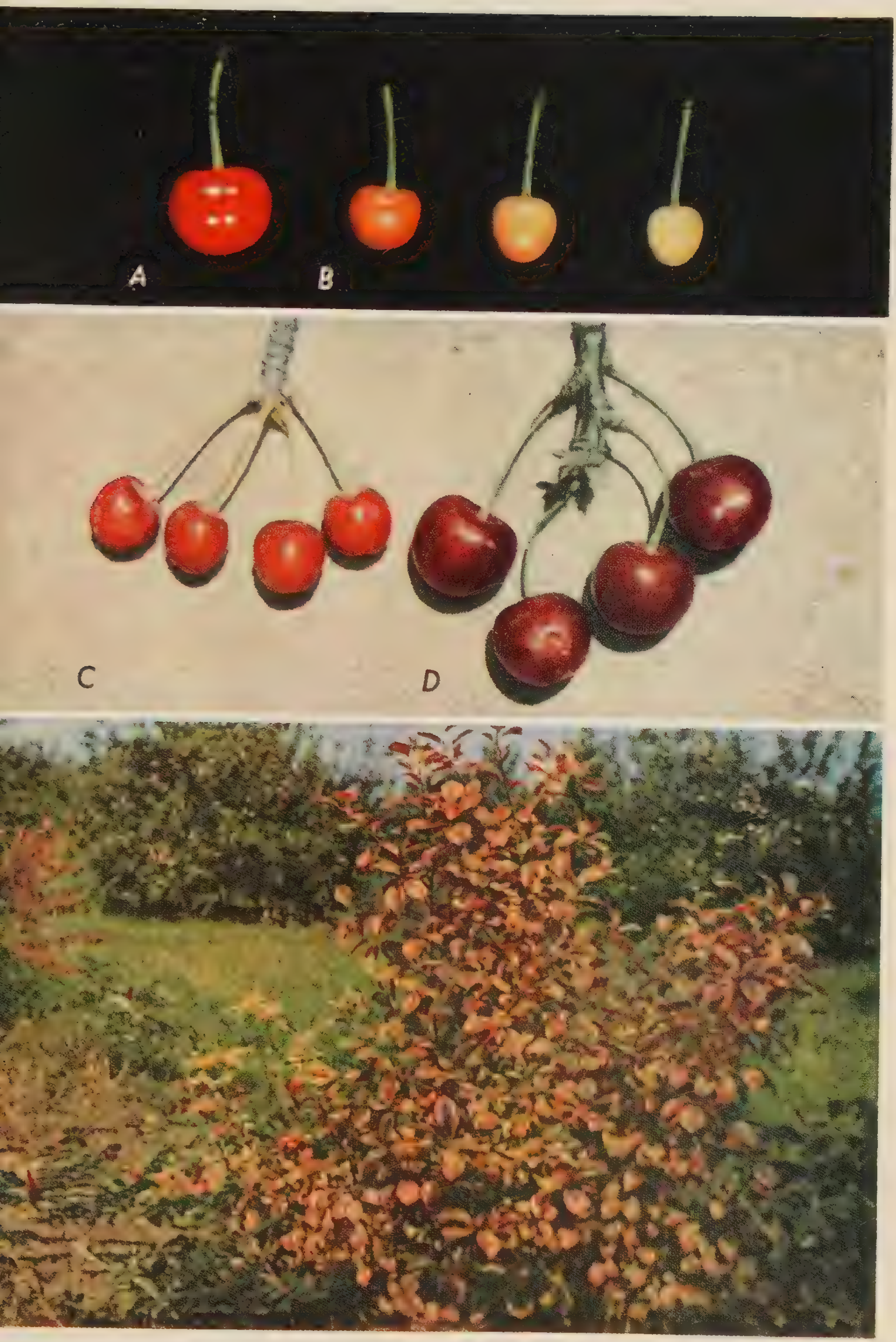
PLATE 4



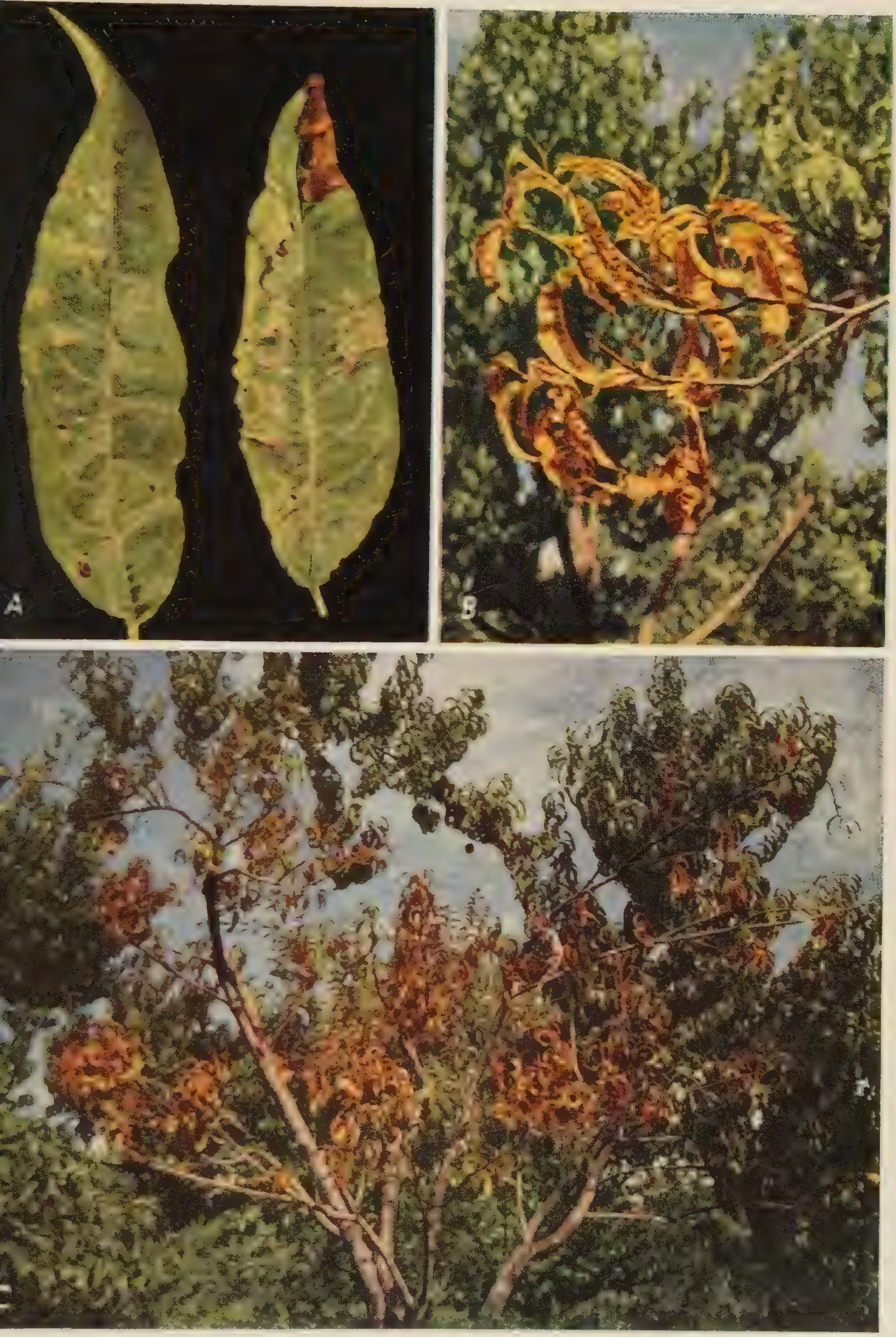
Peach mosaic: *A*, Florence peach flower showing broken color pattern; *B* and *C*, J. H. Hale peach (*B*) affected fruits, showing reduced size, bumpy surface, and uneven color as compared with normal at lower right, and (*C*) leaves showing a gradient of mosaic mottling (veinlet clearing, right; brilliant mottle, center; and marginal striping, left); *D*, partially affected J. H. Hale peach tree, showing retardation on affected side.



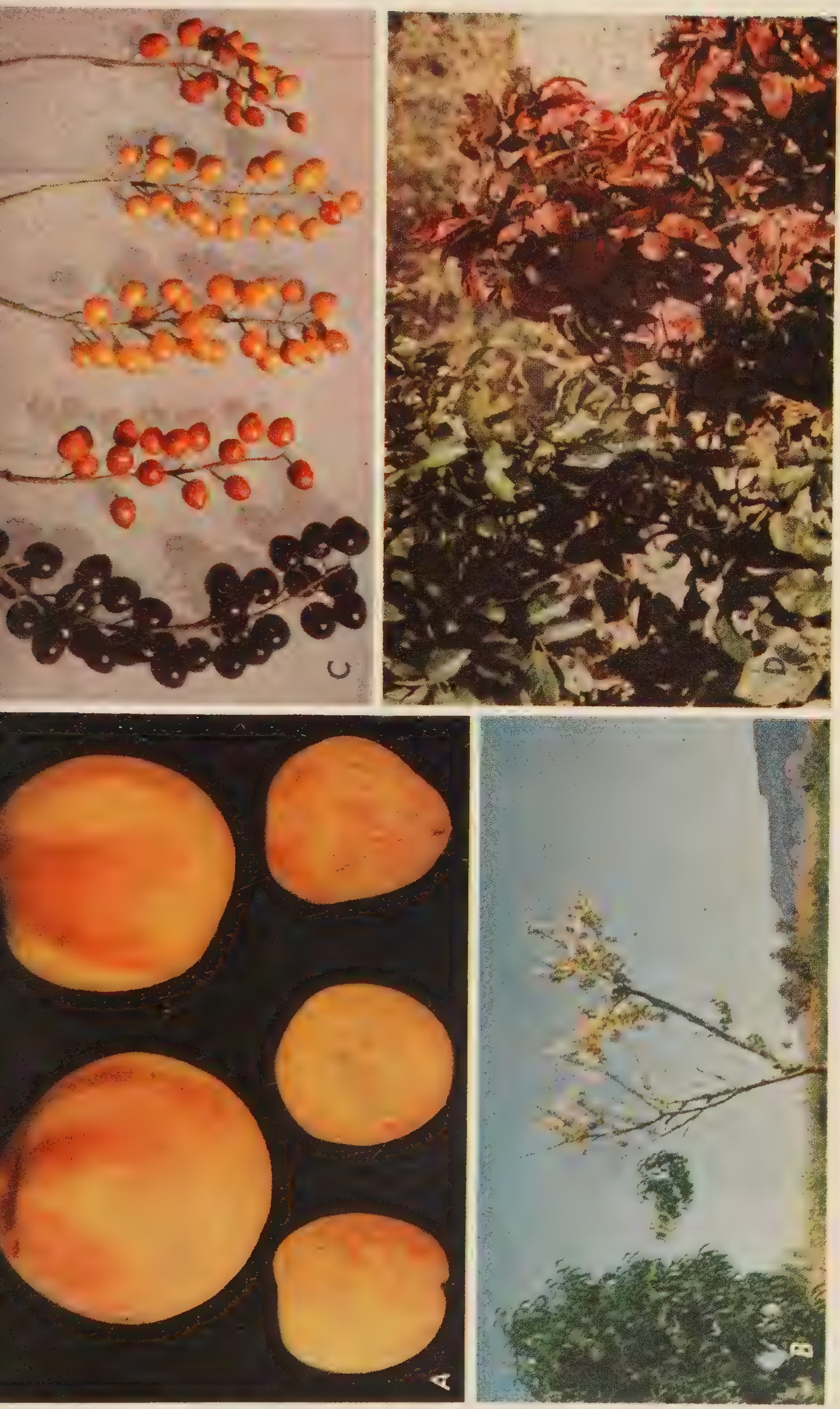
Peach tree naturally affected with X-disease, showing abnormal coloration, leaf rolling, and defoliation of affected branches as contrasted with unaffected branches.



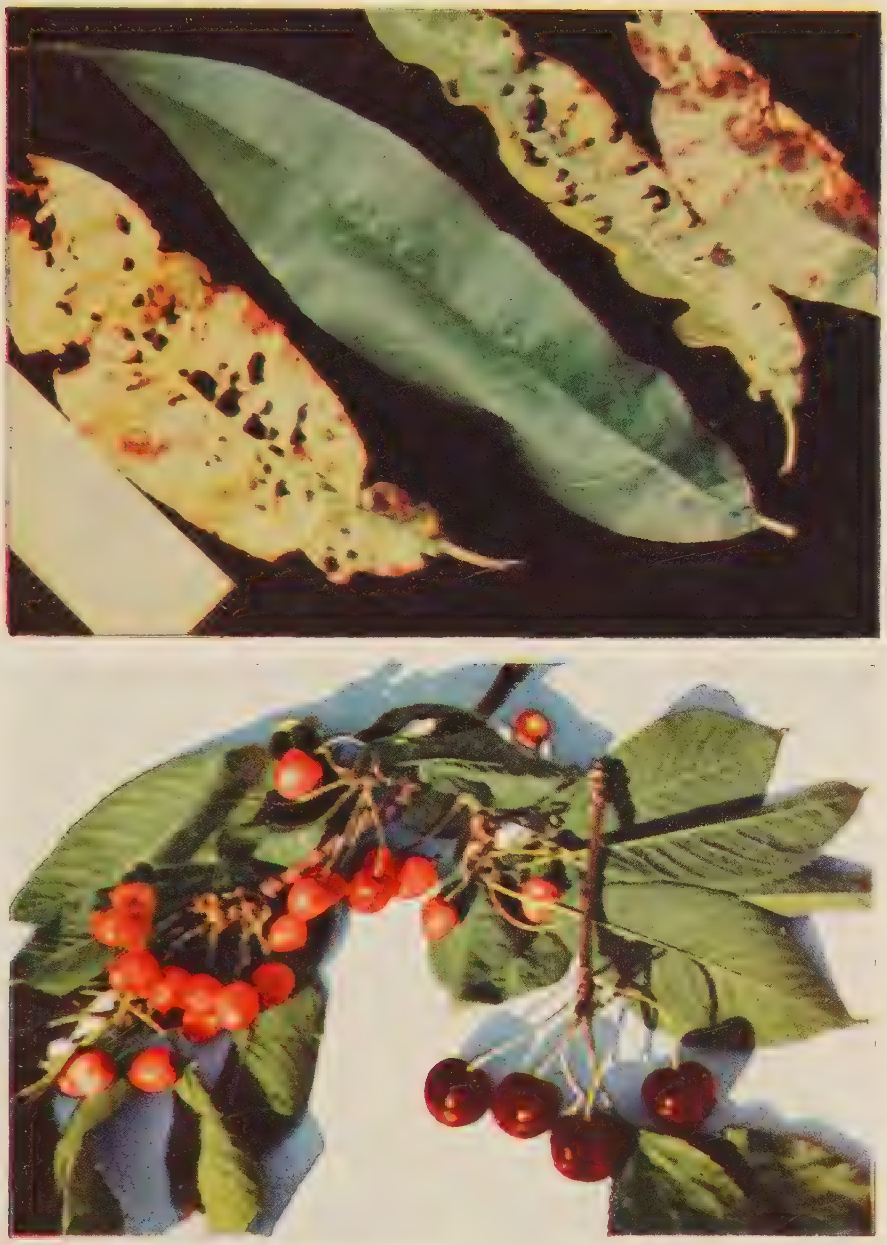
d *B*, Montmorency sour cherries: *A*, Normal; *B*, affected with X-disease. *C* and *D*, Windsor sweet cherries: *C*, Affected with X-disease; *D*, normal. *E*, Chokecherry affected with X-disease, showing brilliant foliage coloration the first season after infection.



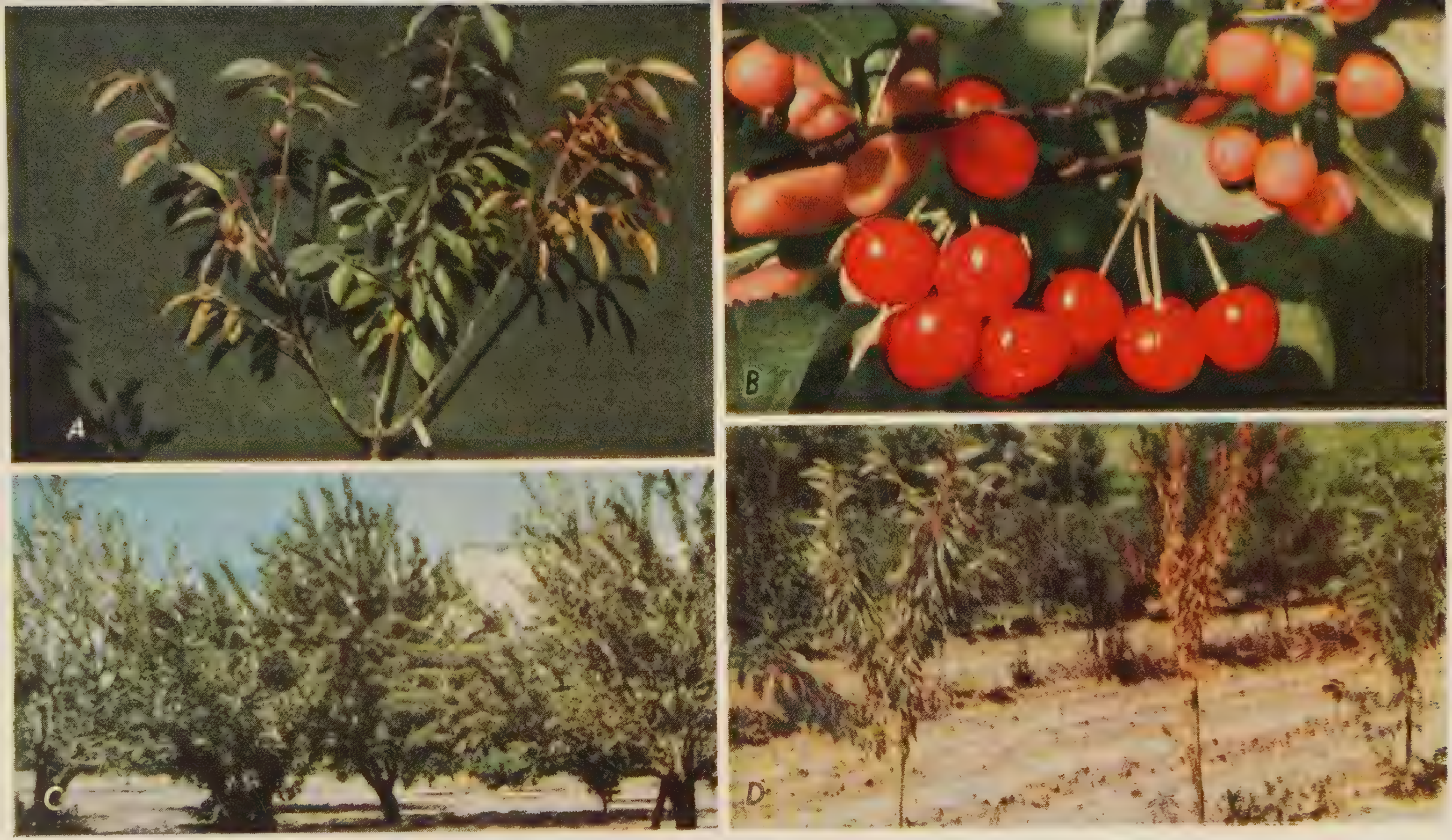
stern X-disease on peach: *A*, Elberta leaves showing early-season symptoms; *B*, Orange Cling branch showing late-season symptoms; *C*, Salwey tree showing affected shoots interspersed with normal ones.



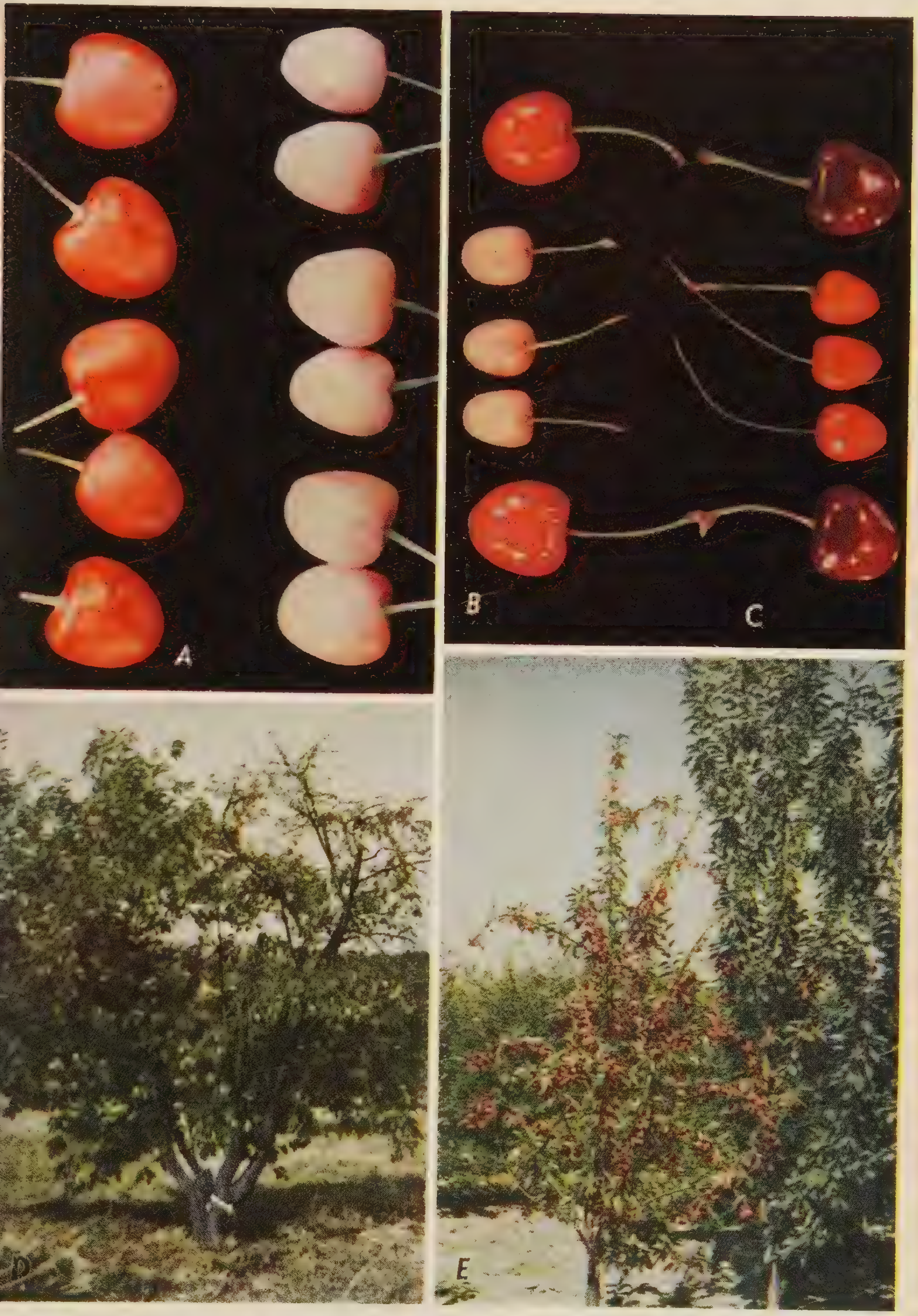
Western X-disease: *A*, Comparable normal and affected Elberta peach fruits; *B*, severely affected Elberta peach tree having only one unaffected branch; *C*, comparable normal and affected western chokecherry fruits; *D*, normal and affected chokecherries showing second-year symptoms.



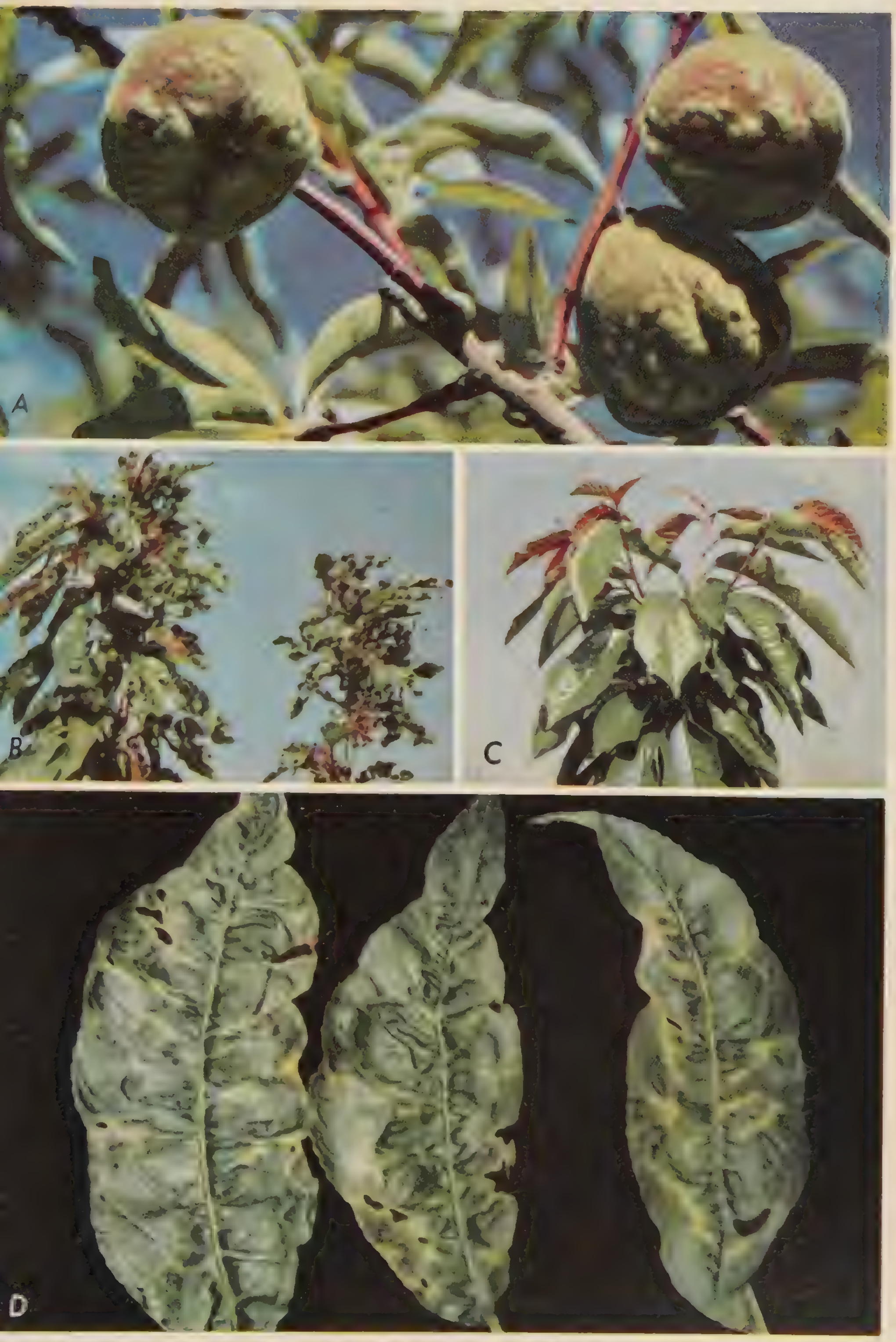
Top: Elberta peach leaves affected with western X-disease, showing greenish-yellow color and irregularly shaped reddish spots, necrotic areas, and holes in contrast with normal leaf. *Bottom:* Normal and western-X-little-cherry-affected Bing cherry fruits.



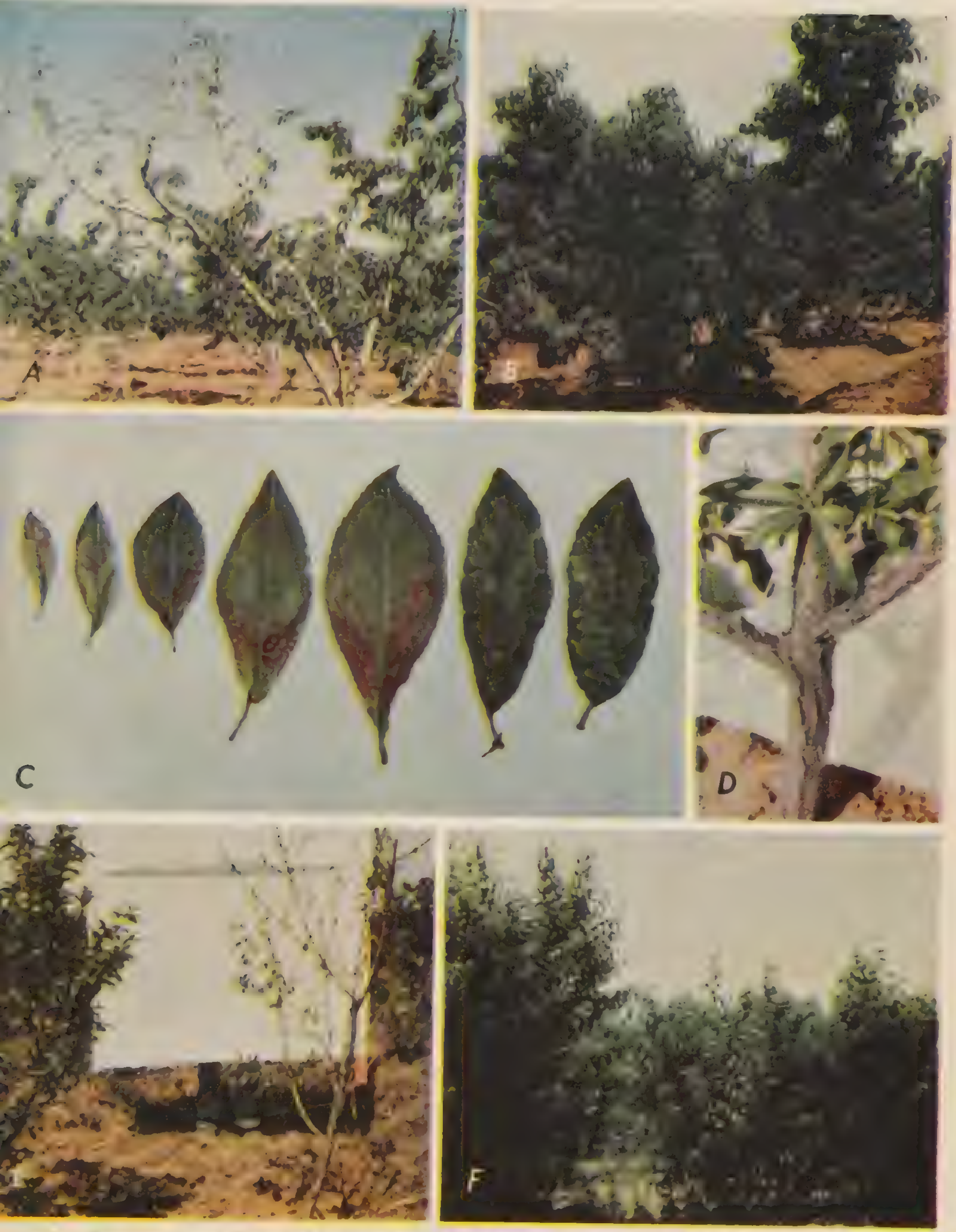
A, Bing cherry top-worked separately on arms of mahaleb cherry understock 2 inches above the crotch; outer branches subsequently inoculated with western X-disease virus. *B*, Normal and western-X-little-cherry-affected Montmorency cherry fruits. *C*, Thirty-year-old Bing tree on mahaleb rootstock naturally affected with western X-disease wilt (right and left). *D*, Young Napoleon cherry trees on mahaleb rootstock inoculated with western X little cherry virus, showing collapse.



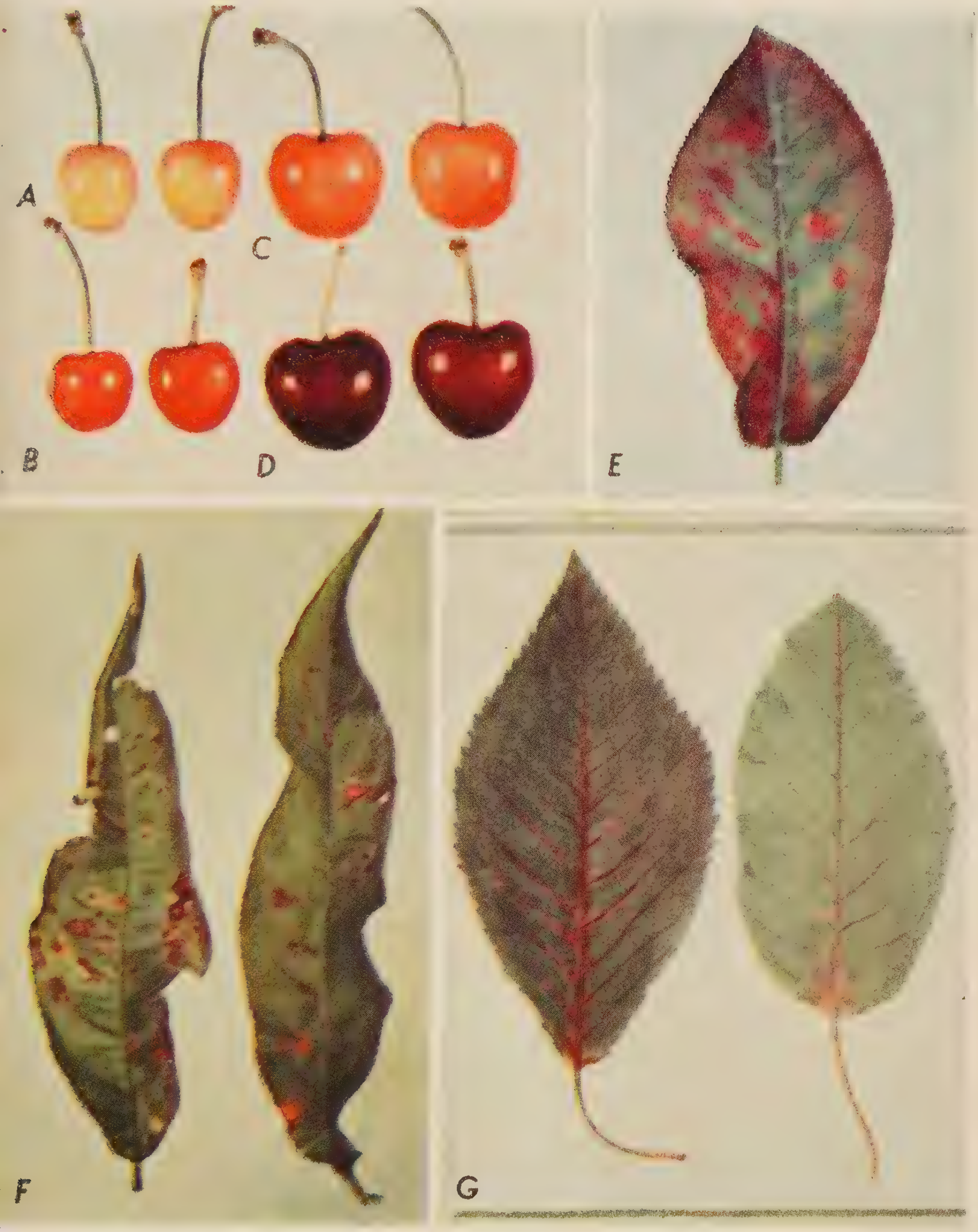
, Normal and western-X-little-cherry-affected Napoleon cherry fruits, Utah; *B*, similar fruits, Washington; *C*, similar Lambert cherry fruits, Washington; *D*, Montmorency cherry tree severely affected with western X little cherry; *E*, 5-year-old Bing cherry tree affected with western X little cherry, showing excessive fruiting.



Peach fruits affected with wart, *B*, shoots of Bing cherry affected with peach mottle, showing mottling and proliferation of leaves; *C*, comparable normal shoot; *D*, peach leaves showing mottling and puckering caused by peach mottle.



Rio Oso Gem peach tree affected with ring spot, showing acute symptoms (retarded foliation, bud killing, and dieback). *B*, Same tree approximately 1 year later, showing recovery. *C–F*, J. H. Hale peach affected with ring spot. *C*, Early spring leaves arranged to show a gradient of symptoms. *D*, trunk of young tree showing bark splitting below inoculation point. *E*, inoculated tree showing shock and dieback in contrast with healthy tree (left). *F*, Three Fay Elberta peach trees pruned to same height the previous winter, showing reduced vigor of two trees chronically infected with ring spot as compared with normal tree (left).



A and *B*, Cherries from buckskin-affected trees: *A*, Napoleon; *B*, Bing. *C* and *D*, Comparable normal fruits. *E–G*, Leaves from plants affected with buckskin: *E*, Western chokecherry, showing carmine color; *F*, peach, showing rolled edges and purple and necrotic spots; *G*, sweet cherry on mazzard rootstock, showing late-summer and fall symptom, orange to red color along the basal portion of the midrib.

YELLOW BUD MOSAIC

By H. Earl Thomas and T. E. Rawlins

Other Common Names of the Disease

Winters peach mosaic.

Name of the Causal Virus

Inops consilii Thomas, Scott, Wilson, and Freitag (*235*).

Geographic Distribution

Yellow bud mosaic has been found in about 50 orchards in Solano and Yolo Counties, Calif.

Economic Importance

Trees of Elberta, Lovell, and Muir peach, the common varieties in the affected district, are reduced in a few years to an unprofitable state; and small trees may be killed in a few months. Almond is injured to an intermediate degree, but it becomes infected infrequently. Apricot is readily infected, but it is damaged little.

Host Range and Varietal Susceptibility

Natural infection is known on almond (*Prunus amygdalus*), apricot (*P. armeniaca*), and peach (*P. persica*) (*233*). Old apricot trees apparently may be symptomless carriers. The peach varieties Babcock, Elberta, Fay Elberta, Florence, Lovell, J. H. Hale, Peak, and Rio Oso Gem are about equally affected when artificially inoculated. Foster is somewhat less susceptible. Under natural conditions Elberta, Lovell, and Muir varieties are seriously injured. The causal virus has been transmitted by grafting to the following species: Almond, apricot, Portuguese laurelcherry (*P. lusitanica*), Japanese apricot (*P. mume*), rose (*Rosa* sp.) variety Ragged Robin, Japanese kerria (*Kerria japonica*), desert peach (*P. andersonii*) on peach roots, sweet cherry (*P. avium*), and myrobalan plum (*P. cerasifera*)—the last by passage from peach through desert almond (*P. fasciculata*).

Symptoms

Peach.—At the onset of disease, pale-green to pale-yellow, oblong, feather-edged blotches develop along the midvein or larger lateral veins of peach leaves, with distortion of the leaf blade and frequent dropping out of the chlorotic areas (fig. 16, *A*). Leaves which are relatively mature at the time of invasion may develop only small chlorotic flecks about 1 mm. in diameter. In advanced stages of the disease the leaves are shed until the basal portions of affected branches may be bare or nearly so (fig. 17). Elsewhere the buds often push out until they are a few millimeters in length and remain practically at a standstill for several weeks (fig. 16, *B*). They are pale yellow. Later such buds die or slowly produce rosettes of small, often distorted leaves with or without mottling. Some dwarfing and malformation may occur on fruits of Muir peach but not on those of Elberta or Lovell. No flower symptoms are known.

Almond.—In the spring the leaves are mottled (fig. 16, *C*) and the foliage is sparse. Later there are terminal tufts of leaves with little or no mottling.

Apricot.—On the leaves there are occasional circular spots, usually about

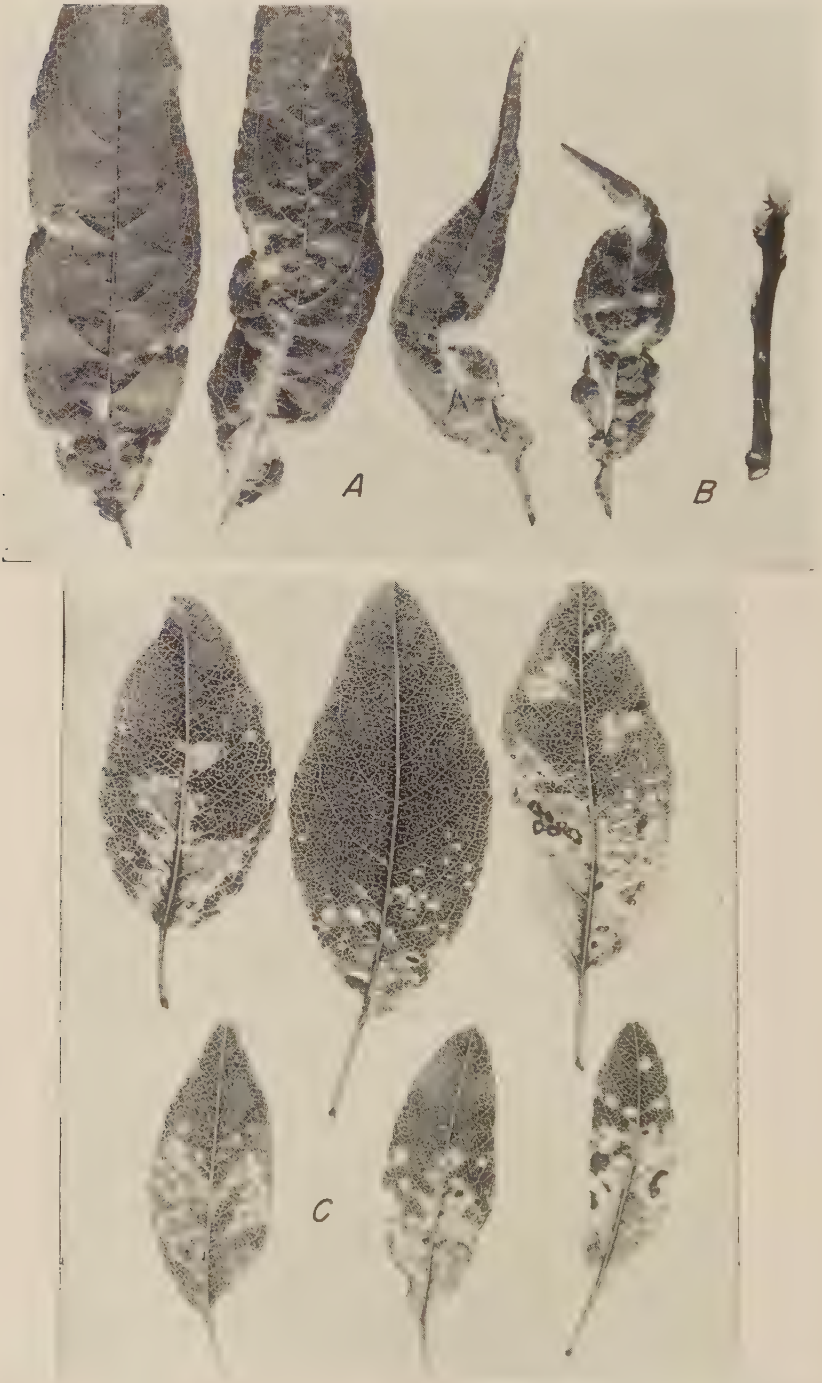


Figure 16.—*A* and *B*, Leaves (*A*) and twig with arrested buds (*B*) of a peach seedling affected with yellow bud mosaic; *C*, leaves of Texas almond naturally affected with yellow bud mosaic, early in season.

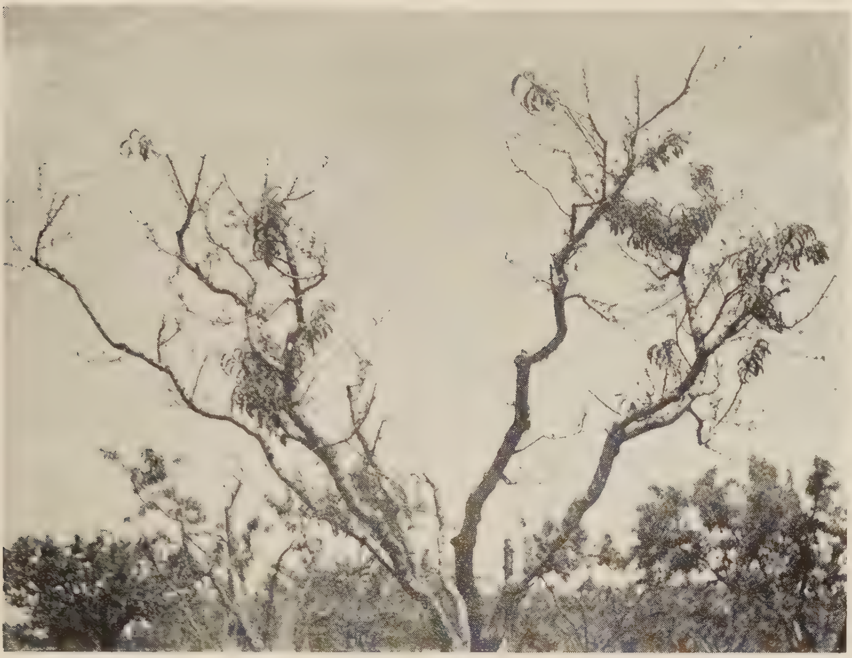


Figure 17.—Orchard Lovell peach tree in advanced stage of infection with yellow bud mosaic.

10 mm. across, with necrotic centers and chlorotic halos. Old trees show no symptoms, though there is evidence that they supply the virus to peach.

Transmission

The disease can be transmitted by grafting. In orchards spread occurs typically from infected trees to adjacent trees only (*235*).

Incubation Period

The incubation period varies from 3 weeks to several months, depending on the growth condition of the tree at the time of inoculation and perhaps on its size.

Control Measures

A quarantine has been placed on the affected district to reduce the movement of infected budwood to other districts. Eradication is being attempted in some orchards, but results thus far are not conclusive. Almond and apricot grow satisfactorily in place of peach but allow the building up of a reservoir of infected material. Pear and plum would be even more sure of success in those parts of the district adapted to them.

WART

By Earle C. Blodgett, J. A. Milbrath, E. L. Reeves, and S. M. Zeller

Names of the Disease

The disease is most commonly called wart. It was referred to as blister, however, in the first report of its occurrence (*13, sec. 3*).

Names of the Causal Virus

The causal virus was named "*Galla verrucae* Blodgett" (*21*). Under the system of naming proposed by Fawcett the name would be "*Prunivir verruca.*"

Geographic Distribution

Wart has been reported as occurring naturally in Adams, Washington, Gem, and Canyon Counties, Idaho (*21*); in Yamhill (*262*) and Malheur Counties, Oreg.; in Chelan, Douglas, Klickitat, and Yakima Counties, Wash. (*184*); and in San Bernardino and Riverside Counties, Calif. (*228*). Infected fruits were observed in 1948 in an orchard in Twin Falls County, Idaho. Artificially infected trees occur in Latah County, Idaho (*21*).

Economic Importance

Generally the number of affected trees is small; but, because of the danger of spread through nursery stock and of possible orchard spread, wart is a serious, potential menace to peach orchards. In 1945 an orchard survey of 300 trees of 1 variety, top-worked in 1941, showed that more than 200 of them bore warty fruits. In this instance all trees of the affected variety were removed (*228*). Since most diseased trees show a high percentage of warty fruits, they are considered useless and also they are hazardous to fruit growing because they are a source of inoculum.

Host Range

Wart is known to occur naturally on seedlings and the Elberta, J. H. Hale, Early Crawford, Candoka, and Halberta varieties of peach (*Prunus persica*) (*21*). It was transmitted by bud inoculation also to Early Crawford, Early Muir, Improved Elberta, Rio Oso Gem, Orange Cling, and Rochester (*262*).

No natural infection is known on sweet cherry (*P. avium*), but Black Republican, Napoleon (Royal Ann), and Lambert varieties were artificially infected by bud inoculation (*262*). Buds from wart-diseased peach trees grafted on young Italian Prune (*P. domestica*) trees produced shoots that bore warty fruit for three seasons, but no symptoms were observed on the leaves or fruits of the prune. Tests have not been completed to determine whether the prune wood carries the virus.

Symptoms

Peach.—The first symptoms appear on the very young fruits, shortly after the calyxes have fallen, as bleached bumps or raised welts on or near the stylar end and often involving half or more of the fruit (*21*, *184*). The fruits are both dwarfed and misshapen if the disease is severe, but slightly affected fruits are nearly normal in size (pl. 12, *A*). The surface of affected tissue varies in color from light tan to conspicuous red and may be rough with warty outgrowths conspicuously raised, or it may be rather smooth or cracked and russeted (fig. 18). Gum is usually present and may be extremely abundant.

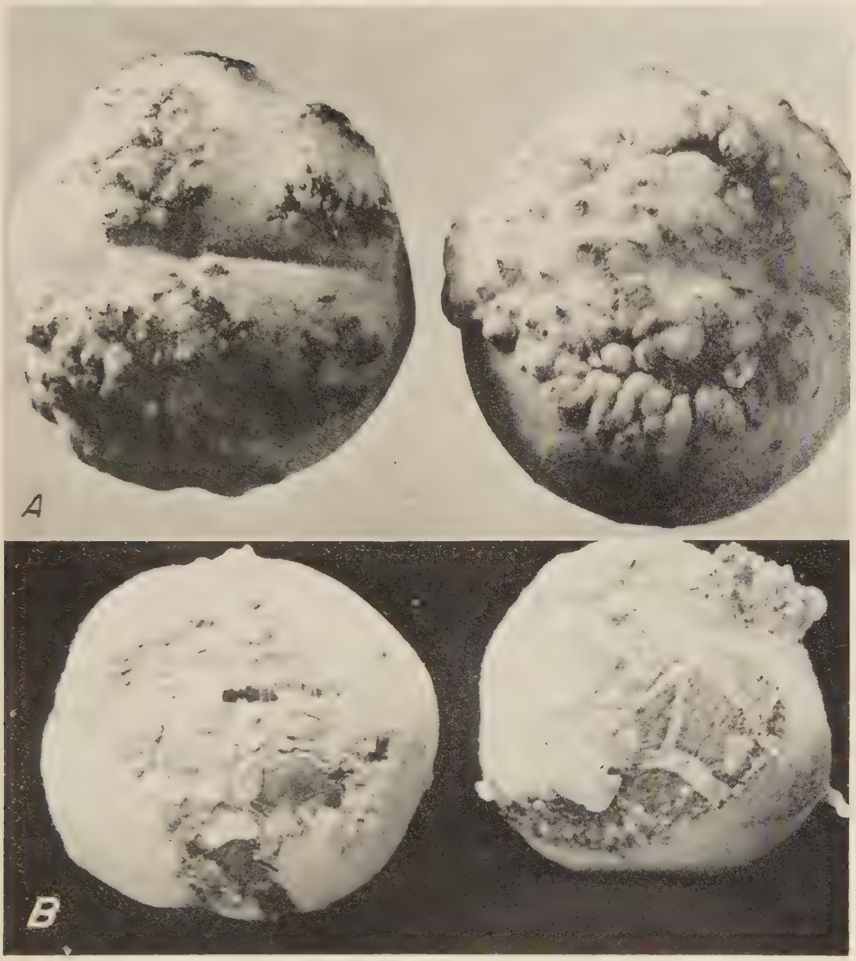


Figure 18.—**Peach fruits with wart: *A*, Rough type; *B*, smooth type.**

The warty tissue is rather superficial, but the underlying tissue is coarse and filled with gum pockets. In some cases the warty tissue is very hard and bonelike, but usually it is tough and leathery. Frequently warty tissue occurs in ringlike patterns near the stylar end.

In the study of peach wart, certain variations were found and described by such names as "smooth wart," "crease wart," and "beady wart." In recent tests by Blodgett it appeared that smooth wart is transmissible as such; that crease wart is not caused by a virus, but is associated largely with certain varieties (Rio Oso Gem in particular); and that beady wart is probably caused by insects. There appeared to be no constant difference in expression of symptoms by the different peach varieties.

In most wart-affected trees observed, there are no leaf symptoms visible. In a few cases, however, there are variable amounts and degrees of mottling, which is often mild and fleeting. This mottling is essentially indistinguishable from that caused by the peach mottle virus (*18, 21*) and has been easily transmitted. Trees having both wart and mottling may, therefore, be infected by virus mixtures.

In general, wart-affected peach trees show no reduction in size, vigor, or production.

Sweet cherry.—Black Republican, Lambert, and Napoleon varieties of sweet cherry showed stem and leaf symptoms when inoculated with peach buds infected with peach wart virus (*262*). The symptoms described by Zeller and Milbrath (*262, p. 607*) are as follows:

> In the Napoleon variety the stem symptoms at first are limited to necrosis in the vascular ring. This necrosis as far as 12 to 15 inches back from the tips results in a general die-back. . . Sometimes there is more than normal branching. In addition the internodes of the last few inches near the terminals of the several branches are shorter and considerably larger in diameter than normal. This condition and resulting "leafiness" of the terminals give a rosetted appearance. There is a tendency toward smaller, narrower leaves as a result of crowding in the rosettes.
>
> * * * * * * *
>
> The mottling produced in the leaves of the Napoleon variety is more characteristic and distinct than that produced in Black Republican or Lambert varieties of cherry. The mottling seems to start with chlorosis bordering veinlets. This spreads out to form larger ring-like patterns or even somewhat rectangular bands or lines on either side of the veins. . . In May and June the symptoms are especially prominent on most of the young leaves. As the season progresses and the leaves mature the symptoms become less conspicuous, until finally in late August the mottling has disappeared in all of the matured leaves, even though they may have been mottled earlier.
>
> There is a similarity between mottling produced in cherry leaves by peach-wart virus and that produced by mild-mottle-leaf virus. Warty fruits, however, are not produced when peach trees are inoculated with mild mottle leaf of cherry. Cherry trees with the mild-mottle-leaf disease likewise do not have the stem symptoms described for peach wart in cherry.

Diagnostic or Unusual Characteristics

Peach wart virus produces spectacular symptoms on peach fruits, but affects the trees very little. One of the diagnostic symptoms is the localization of wart tissue near the stylar end of mildly affected peach fruits or its prevalence there on severely affected fruits.

Transmission

Artificial infection by peach wart virus has been obtained principally by budding or inarching. It has been shown that severity of wart symptoms in one season after inoculation is related to the amount of inoculum used (*21*). Zeller and Milbrath obtained severe infection in 4-year-old peach trees by the insertion of three to five diseased buds. Inoculations made by Blodgett in 1943 showed by 1945 that transmission of the virus was easily obtained by grafting warty fruit tissue into young peach tree trunks (*26*). Wart was transmitted by Zeller and Milbrath from peach to sweet cherry and back to peach by bud inoculation (*262*).

Incubation Period

Diseased peach buds inserted September 7, 1940, produced warty fruits on some peach trees by June 13, 1941, whereas in other cases evidence of transmission was not visible until the second season. Sweet cherry trees bud-inoculated in August began to show leaf and stem symptoms the next April (*262*).

Control Measures

It is recommended that all diseased trees be removed promptly and that budwood from wart-free trees be used for propagation.

PEACH MOTTLE

By Earle C. Blodgett

Other Common Names of the Disease

None.

Name of the Causal Virus

Peach mottle virus.

Geographic Distribution

Single naturally infected peach trees were found in Gem and Payette Counties, Idaho, and several experimentally diseased trees are known in Latah County. On the basis of symptoms on one tree seen by the author the disease is considered to occur in Malheur County, Oreg. A mottle associated with peach wart was seen in Canyon County, Idaho. (See *13, sec. 2; 17; 19; 20; 21.*)

Economic Importance

Peach mottle is regarded as of minor importance at present.

Host Range

The disease has been seen in orchards only on Elberta peach (*Prunus persica*). In experimental-transmission tests mottle symptoms were produced on the varieties J. H. Hale, Elberta, and Slappey and on peach seedlings; on sweet cherry (*P. avium*) varieties Bing, Napoleon (Royal Ann), Lambert, and Black Republican and on mazzard (*P. avium*) seedlings; on sour cherry (*P. cerasus*) variety Montmorency; and on May Duke (semisweet cherry). The Moorpark apricot (*P. armeniaca*) is listed as a possible host of peach mottle, but it has been inadequately tested. In several inoculation trials no definite symptoms were produced on the Italian Prune variety of domestica plum (*P. domestica*). No tests were made to determine whether this variety is a symptomless carrier.

Symptoms

Peach.—The original affected Elberta tree presented a ragged appearance and in general was lighter green than normal trees. Growth was reduced, but rosetting was not characteristic. The fruits showed no consistent differences from normal, and no breaking in the color pattern was observed on the blossoms. The leaf symptoms consist of coarse mottling, slight dwarfing, rolling of the margin, deformity, and puckering (fig. 19, *A;* pl. 12, *D*). Leaf symptoms on the different peach varieties are similar, but the amounts of mottling vary markedly. Young trees bud-inoculated with peach mottle virus grew poorly, and several died. Symptoms are pronounced early in the season; later they become partially masked.

Cherry.—The first tests on transmission of peach mottle to Bing cherry resulted in very marked symptoms, and at first peach mottle was thought to be similar to cherry mottle leaf (*183*) (fig. 20, *C, D*). Later, however, much more extensive dwarfing, mottling, and necrosis of foliage accompanied by marked tree stunting, shoot necrosis, and dieback showed the disease to be distinct from mottle leaf (pl. 12, *B*). Symptoms on the Napoleon variety are similar to those on Bing except that the effect is generally much less severe. Inoculated trees of the Lambert variety developed a chlorotic veinlet banding

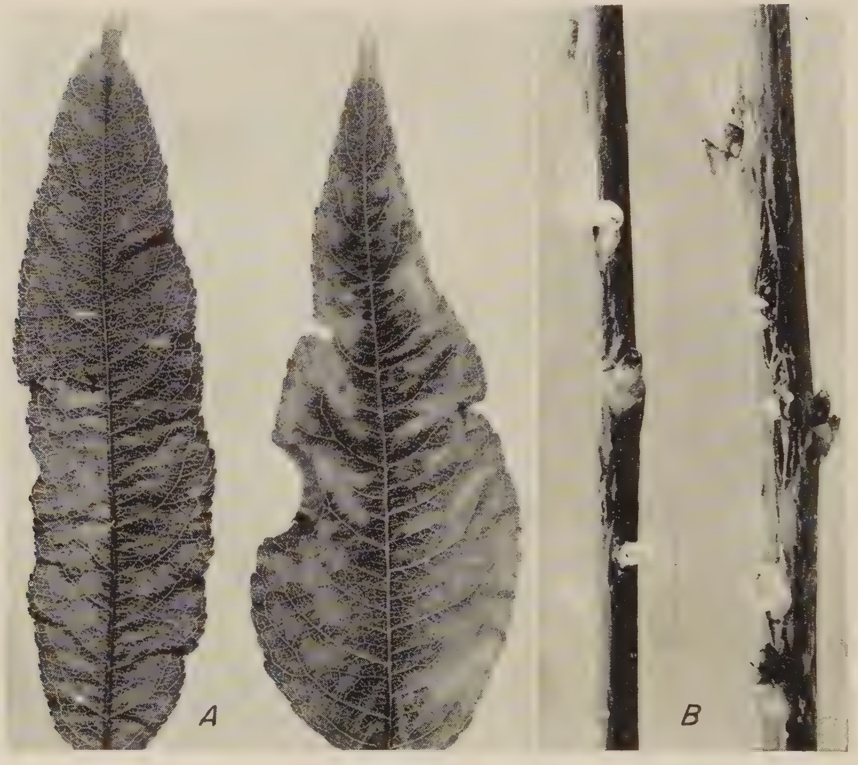


Figure 19.—A, Leaves from a J. H. Hale peach tree artificially infected with peach mottle virus; B, twigs from a Montmorency cherry tree affected with peach mottle, showing gum exuded from pockets in the bark.

appearing much like a golden-net pattern. Symptoms on the Black Republican variety (fig. 20, *A*) are similar to those on Lambert, but they are much more severe, giving the foliage a light-green color. This reaction, similar to that on Lambert, distinguishes cherry mottle leaf from peach mottle. Symptoms of mottling developed on inoculated mazzard seedling stock and on mazzard shoots from roots on infected Bing trees.

On the Montmorency variety of sour cherry peach mottle produces mild to severe mottling (fig. 20, *B*) and much twig dieback. Associated with this dieback is an abundant formation and exudation of gum on both old and young twigs (fig. 19, *B*). In some cases the gum pockets cause stem swellings, cracks, or both in the bark. There is a tendency for trees to appear somewhat rosetted.

Diseased trees of the May Duke variety are dwarfed, principally from the necrosis and dieback of terminal shoots. Pronounced leaf mottling and vein clearing also are present.

Apricot.—In one test with two Moorpark apricot trees, definite, although mild, mottling of the foliage appeared the next season after bud inoculation. No transmission test was made from these trees back to cherry or peach to determine whether the symptoms on apricot were a result of the peach mottle virus.

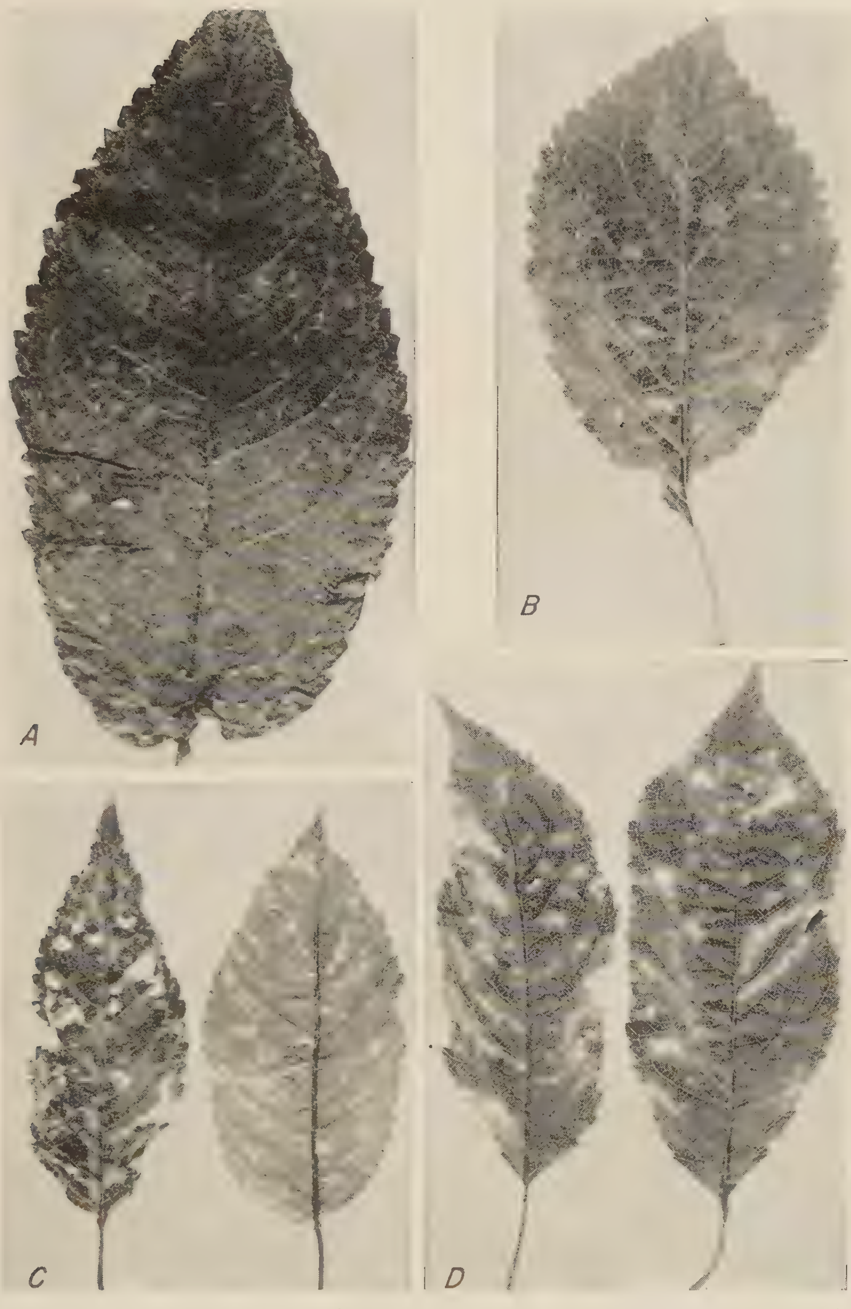


Figure 20.—A–C, Leaves from cherry trees artificially infected with peach mottle virus: *A,* Black Republican; *B,* Montmorency; *C,* Bing. *D,* Comparable leaves from Bing cherry tree affected with mottle leaf.

Diagnostic or Unusual Characteristics

Peach mottle virus produces severe symptoms on Lambert, Black Republican, and Montmorency cherries; therefore, it is distinguished from cherry mottle leaf, which produces only mild symptoms on these hosts. In tests made by Cochran [15] in Texas with Idaho peach mottle material, the virus was determined to be distinct from that causing peach mosaic although on peach both produce mottle symptoms which are similar in some respects. Peach mottle is further distinguished from peach mosaic by the production of symptoms on cherries, which are immune from the peach mosaic virus.

Transmission

Repeated trials have shown that peach mottle is easily and consistently transmitted by means of buds from peach to peach and from peach to cherry (both sweet and sour) and back to peach (*18*).

Incubation Period

Peach or cherry trees bud-inoculated in the fall (September) usually show symptoms of mottle when they leaf out the next spring.

Control Measures

Infected trees should be removed promptly, and budwood from healthy trees should be used for propagation.

Remarks

In some peach trees affected with wart a mottling of the leaves similar to that produced by peach mottle was noted. Transmission from such trees resulted in both wart and mottle on peach and a reaction on cherry similar to that caused by the peach mottle virus. Such cases undoubtedly represent infection by at least two viruses.

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[15] Cochran, L. C. Unpublished data.

MUIR PEACH DWARF

By Lee M. Hutchins, C. F. Kinman, L. C. Cochran, and Gilbert L. Stout [16]

Names of the Disease

"Muir peach dwarf" is proposed as a good descriptive name of the disease for reasons that appear under Symptoms. The term "rosette" was employed in the earliest mention of the disease by the late W. T. Horne in 1920, and the name "Muir rosette" has been used *(213, p. 91)*.

Name of the Causal Virus

Muir peach dwarf virus.

History and Geographic Distribution

The disease here called Muir peach dwarf came to Kinman's attention in an old orchard of the Muir peach variety near Chico, Calif., in September 1935. Affected trees exhibited symptoms suggesting phony, and Hutchins, who had worked extensively with that disease in Georgia, was requested to examine the trees near Chico. This led to the investigational work that yielded the information presented here.

Upon approaching the affected Muir peach trees, the investigator noted the very striking resemblance of the disorder to phony. Closer observations, however, disclosed points of difference. Also the chemical test for phony (pl. 3, *D*) was clearly negative when made on roots of Muir trees showing advanced stages of the disorder, and the latter was regarded as a new disease.

The information obtained during these examinations and photographs of affected trees were brought to the attention of Max W. Gardner and Ralph E. Smith, Department of Plant Pathology, University of California, Berkeley. They recalled earlier mention of apparently the same disorder and referred to records left in the Department by Horne. With the kind permission of Gardner, these first-known records of Muir peach dwarf are summarized here.

On April 30, 1920, Horne observed on Muir peach trees at Suisun Valley, Solano County, symptoms which he regarded as resembling peach rosette or phony; the grower stated that the trees had been affected for several years. Horne further noted that Elberta peach trees interplanted with the Muir trees in this orchard were not affected. On October 28, 1920, he observed the same disorder at Mountain View, Santa Clara County, where 2 or 3 acres of Muir trees showed shortened twigs. Apparently the trouble had been present for 10 or 12 years and had spread windward. Smith supplemented these statements with the interesting comment that, in passing the orchard at Suisun Valley about October 10, 1935, he observed that the disease was still there.

With the discovery of extremely dwarfed Muir trees at Chico, about 150 miles north of Suisun Valley, the disease assumed new interest and it seemed desirable to determine whether it was infectious. The results of inoculation experiments performed by Hutchins and Kinman in 1936 and 1937 placed Muir peach dwarf in the group of graft-transmissible virus diseases. Various phases of the work were further developed by these investigators until July

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[16] By mutual agreement associate authorship is not alphabetical.

1941. Since 1941 Cochran and Stout have continued with the research and observations.[17]

Muir peach dwarf has been seen only in orchards of the Muir peach variety and is known to occur in several counties of middle California. It has not been reported elsewhere.

Economic Importance

Muir orchards lightly infected or free of the disease may adjoin or be near heavily infected orchards. Spread tends to be local, slow, and mostly in close-colony formation. Mature fruits on diseased trees are predominantly of good size and quality except where these are in dense, unthinned clusters at the base of short whorls issuing from branch terminals of the previous year. With increasing age of the disease in the tree, annual growth is greatly reduced, branch terminals tend to die back, the crop is progressively less from year to year, and in old cases the crop may be almost a total failure.

Muir is a superior drying variety and an excellent dessert peach. In 1946 there were 6,110 acres of Muir orchards in California. In acreage the variety was third of the freestones and comprised about 10 percent of the freestone peach acreage in the State. Progressive losses from Muir peach dwarf are known to have been heavy in the Santa Clara Valley and in some other sections. Muir peach dwarf may have been one of the factors that discouraged planting of the Muir variety in recent years. From 1937 to 1946, inclusive, only 395 acres of Muir plantings were made in California.

Host Range and Varietal Susceptibility

In nature Muir peach dwarf has been noted on only the Muir variety of peach (*Prunus persica*) and on no other stone fruits. By graft inoculation with buds from diseased Muir orchard trees the Muir peach dwarf virus was successfully introduced into nursery trees of several peach varieties, on which relative injury was as follows: Severe on Muir, Paloro, and Alexander; medium severe on Lovell; slight on J. H. Hale; and absent on Elberta. It was also introduced by graft inoculation into nursery trees of the following stone fruits, all of which proved to be symptomless carriers: Nonpareil almond (*P. amygdalus*) on almond rootstock, Royal apricot (*P. armeniaca*) on apricot rootstock, Napoleon (Royal Ann) cherry (*P. avium*) on mazzard (*P. avium*) rootstock, mahaleb cherry (*P. mahaleb*) seedlings, and French (Agen) prune (*P. domestica*) on myrobalan plum (*P. cerasifera*) rootstock. The virus was introduced also into French prune and an unidentified variety of almond, without producing symptoms, by top-working them on Muir peach trees severely affected with Muir peach dwarf.

Symptoms

The most striking symptom of Muir peach dwarf is to be seen in the growing season, when the trees are in full leaf. The dwarfed, diseased trees (fig. 21, *A*) show a profusion of large, flattish deep-green leaves, rather closely appressed on short twigs. By comparison, in summer a normal Muir tree (fig. 21, *B*) displays lighter green leaves that are curved along the midrib and are evenly disposed on naturally spaced, long twigs.

Shoot characters, apart from the foliage, are best seen in the dormant season. Affected trees (fig. 22, *A*) show a marked tendency to produce a whorl of three to eight new shoots from branch terminals of the previous sea-

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[17] None of the results have been published. Therefore, the present account is given in more detail than otherwise would be required.

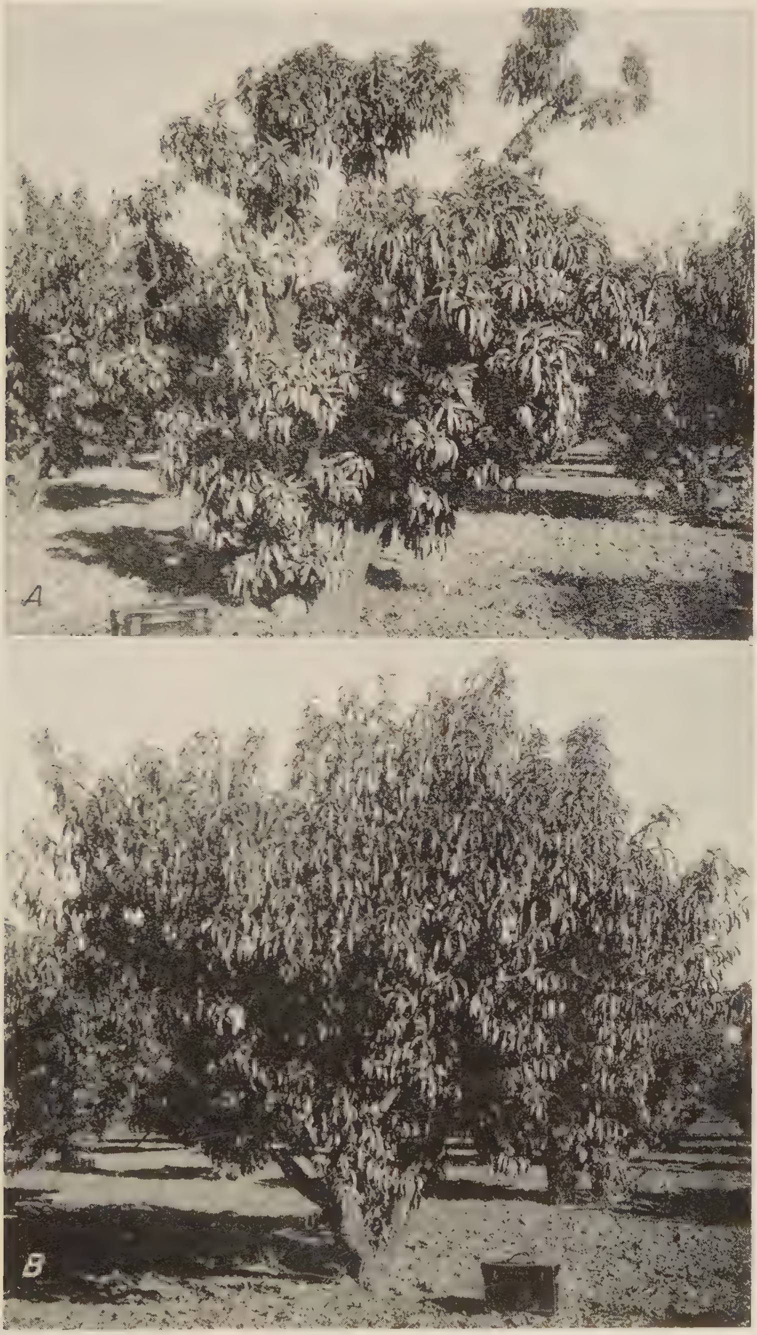

Figure 21.—*A*, Muir peach tree severely affected with Muir peach dwarf, with most of the branch terminals, which had weakened and died back, pruned off, showing large flattish leaves and a dense foliage mass at base of tree; *B*, comparable unaffected tree.

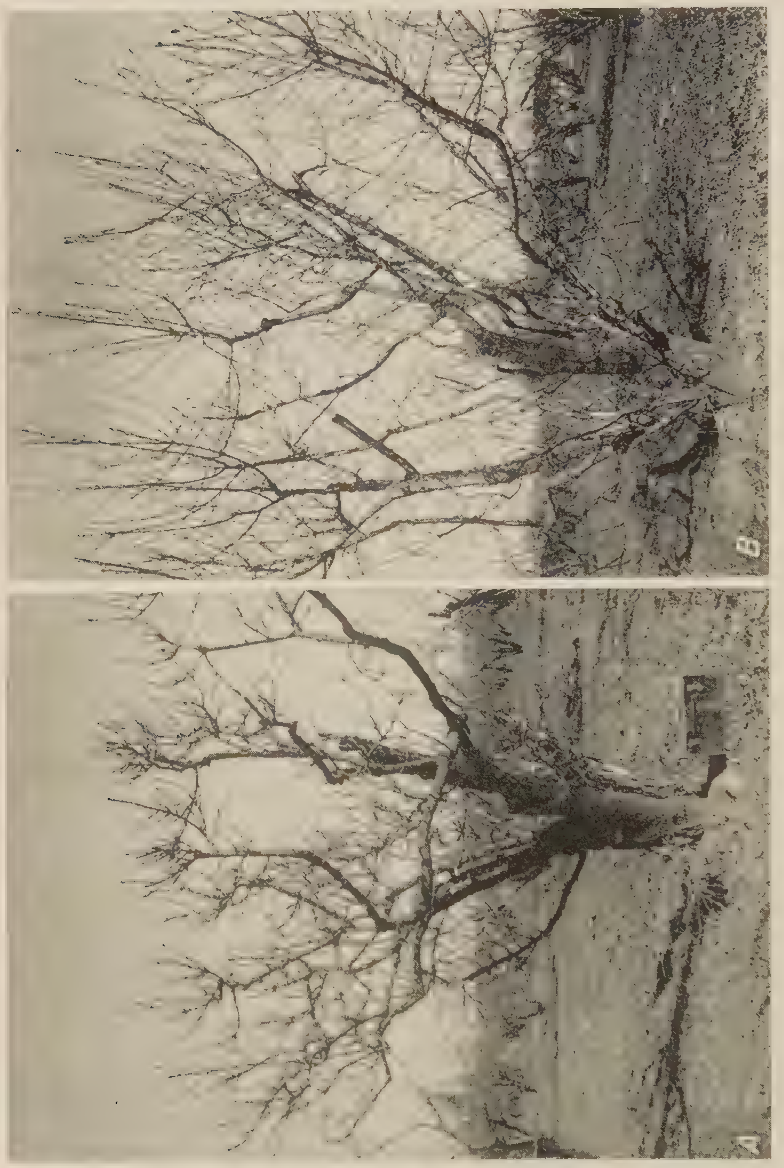

Figure 22.—*A*, Old Muir peach tree severely affected with Muir peach dwarf, showing branch and twig characteristics; *B*, comparable unaffected tree.

son's growth. Lateral twigs are dwarfed and more closely spaced along the extending limbs than is the case on a normal tree (fig. 22, *B*).

After some years of the disease, scattered branch terminals weaken or die, the dying sometimes extending downward along the limbs for a few feet. When such limbs are pruned off, they are not replaced by new, upward-growing shoots. Instead, the pruned trees are apt to produce a quantity of short, branched, heavily leaved growth from the framework limbs; and sometimes they produce suckers from the root collar. Such growth produces a very heavy deep-green foliage mass about the base and framework arms of the tree.

Owing to the short internodes, the flowers on most of the diseased twigs (fig. 23, *A*) are much closer together than on normal twigs (fig. 23, *B*). In general, the internodes are apt to be extremely short for an inch or two, after which they are somewhat longer and on some twigs may attain the normal length. Frequently, at the end of the growing season the current season's growth is considerably thickened and branched and may have extremely short internodes, from which springs a whorl of twigs the following season (fig. 24, *A*). The short, diseased twigs are usually thickened throughout their length and are apt to be pale green as compared with the reddish-brown color of normal twigs. Failure to develop the brown color is probably due to dense shade from the compact, heavy foliage.

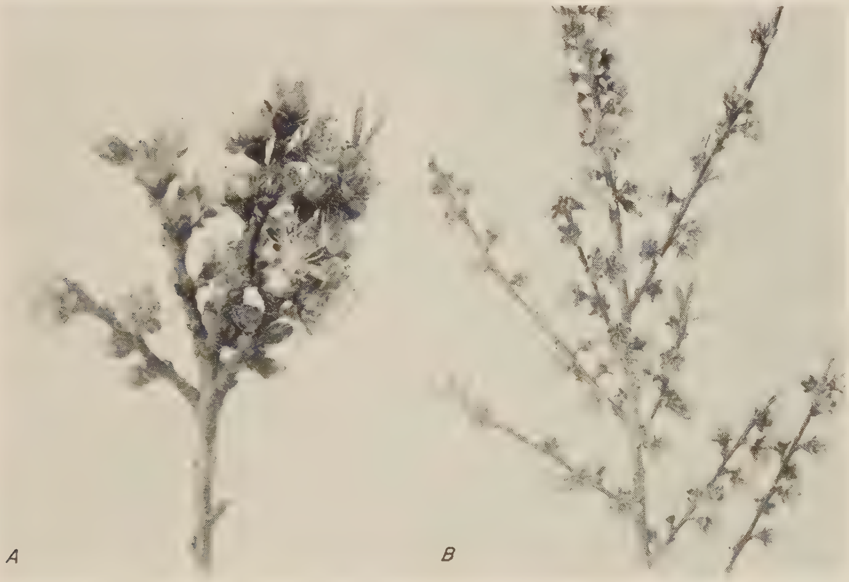

***Figure 23.*—*A*, Branch of Muir peach affected with Muir peach dwarf showing twig, bud, and flower characteristics; *B*, comparable unaffected branch.**

The leaves from basal buds of diseased twigs are usually smaller than normal, but are well formed. Leaves along the greater portions of the twigs are as large as normal leaves or larger. Near the terminals of the season's twig growth, the leaves on affected trees are not quite full size and tend to be slender. All leaves on diseased trees are of a uniform green color; they show no spots, mottling, or deformities whatsoever. They may remain green after

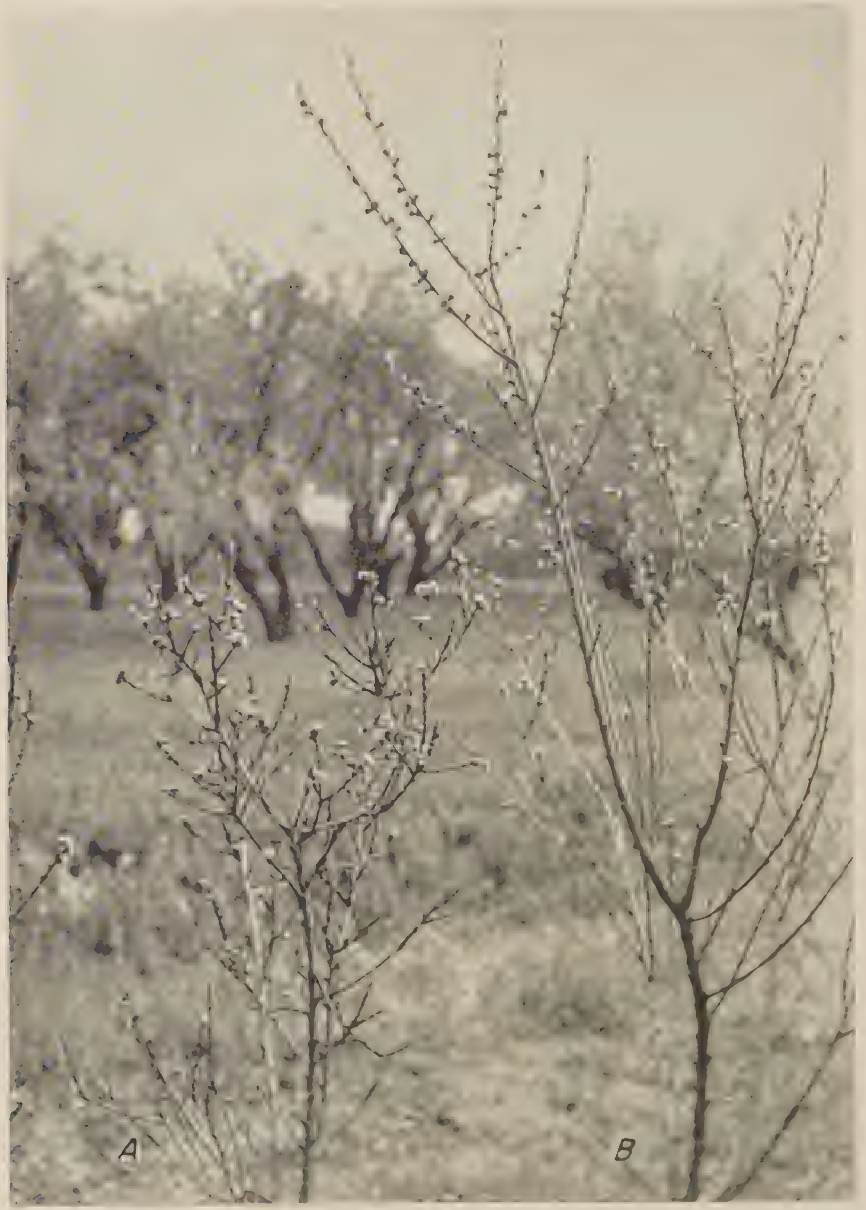

Figure 24.—Muir peach nursery trees of the same age, showing the stunting effect of Muir peach dwarf: *A*, Diseased, 3 years after inoculation; *B*, unaffected.

normal foliage takes on autumnal coloration. Flowering and leafing in spring may be slightly delayed on diseased trees.

Fruits on diseased trees ripen about the same time as normal fruits, and the peaches may be of good size and quality if the trees are not too severely dwarfed. On the most severely diseased trees on which dying arms have been cut back heavily, the peaches are large and misshapen, with a tendency to develop split pits. Fruit production is greatly reduced with increasing age of the disease, and in the most advanced cases there may be only a dozen fruits per tree.

Transmission and Natural Spread

Laboratory cultures failed to reveal the presence of a bacterial or fungus parasite in the living tissues of affected trees. The disease was readily transmitted by grafting roots, bark, scions, or buds from diseased trees to the roots or the shoots of normal Muir peach trees. Therefore Muir peach dwarf was attributed to an infective virus.

Presence of the Muir peach dwarf virus in the varieties or species of peach, almond, apricot, cherry, and plum graft-inoculated (p. 64) was determined by budding back to uninfected Muir seedlings, which developed typical symptoms of Muir peach dwarf. Although the French prune and the unidentified variety of almond top-worked on Muir trees severely affected with Muir peach dwarf made extensive normal-appearing growth, buds from such growth when indexed on uninfected Muir seedlings also induced typical symptoms of Muir peach dwarf on them.

The natural vector of Muir peach dwarf virus has not been determined. Natural spread of the disease was demonstrated in the Muir variety by annual surveys in individual orchards.

Attempts were made by Cochran and Stout to recover Muir peach dwarf virus from symptomless orchard trees of several sorts growing near heavily diseased Muir orchards. At Morgan Hill, Calif., a block of old Muir trees almost 100-percent-affected with Muir peach dwarf was bordered by a row of healthy-appearing old Elberta trees. Muir peach dwarf spreads in close-colony formation, and it would be expected that some of the Elberta trees would be carrying the virus even though they showed no symptoms of the disease. Each of the Elberta trees in the row immediately adjacent to the diseased Muir trees was indexed by budding on normal Muir nursery trees. None of the index trees developed the disease, and it was concluded that at least in such locations as the one investigated the hazard of natural spread of Muir peach dwarf to Elberta trees was negligible.

Healthy-appearing old almond trees in an orchard row adjacent to a heavily diseased Muir peach orchard at Chico were indexed for the presence of Muir peach dwarf virus. None of the Muir index trees developed the disease. Similarly, old orchard trees of apricot and French prune adjacent to severely diseased Muir orchards were indexed. In no instance did the Muir index trees develop Muir peach dwarf.

Under maximum locational opportunity for natural spread of Muir peach dwarf, the causal virus did not find its way from heavily diseased Muir orchards to adjacent orchards of Elberta peach, almond, apricot, and French prune. Nevertheless, all of these sorts are known to be symptomless carriers of Muir peach dwarf virus if the latter is introduced by graft inoculation. It has been shown that some peach varieties other than Elberta will develop symptoms of Muir peach dwarf following graft inoculation and that symptom manifestation will vary from mild to severe, depending upon the peach variety. However, natural occurrence of Muir peach dwarf on any of these peach varieties has not been reported.

Within the limits of the present knowledge of Muir peach dwarf as it occurs in nature, this very interesting and unique virus disease appears to be confined to the Muir peach variety and it has not been reported outside California.

Incubation Period

As determined by graft inoculations the incubation period of Muir peach dwarf is less than 1 year. When diseased scions were grafted on healthy Muir peach orchard trees in winter, symptoms began to develop in the vicinity of the inoculum the following spring. Normal Muir nursery trees grafted in summer with buds or bark from diseased trees developed symptoms of Muir peach dwarf the next growing season.

Control Measures

Propagating material of the Muir peach variety should be selected at a considerable distance from diseased Muir trees, and preferably it should come from districts entirely free of the disease.

In orchards only trees of the Muir peach variety are known to contract Muir peach dwarf; diseased trees are very easy to identify; spread is slow and occurs predominantly in close-colony formation; and the incubation period, as determined by graft inoculation, is less than one year. Therefore, under such favorable conditions for rapid detection of infected trees, well-directed eradication procedures should be very effective in controlling the disease.

RING SPOT

By L. C. Cochran, Lee M. Hutchins, J. A. Milbrath, Gilbert L. Stout, and S. M. Zeller

Names of the Disease

Ring spot was originally described on peach (*62*). The name was applied because ring and spot symptoms are consistently produced on peach and most other host plants. Probably the first record of the disease was made by Valleau (*242*). In working with yellows and an unidentified trouble on plums, he described symptoms on inoculated peach seedlings which indicate the presence of forms of the ring spot virus in the complex. The disease was probably also encountered by Thomas and Rawlins (*233*) in work with the buckskin virus and recorded as a mosaic of mazzard cherry. Later publications on what may be the same virus, or forms of it, refer to a disease produced on sour cherry as necrotic ring spot (*91, 163, 164*) and to one on sweet cherry as lace leaf (*256*).[18] It is possible that tatter leaf on sweet cherry described by Willison and Berkeley (*249*) may also be caused by the ring spot virus. The production of symptoms on midsummer leaves of trees affected with necrotic leaf spot of peach (*49*) differentiates this disease from ring spot, which produces symptoms only on leaves formed in the spring.

Names of the Causal Virus

No Latin binomial has been applied to the causal virus except that the virus causing ring spot on sour cherry has been referred to as *Annulus cerasae* by Hildebrand (*91*). Under the naming system proposed by Fawcett, the name would be "*Prunivir circummaculum*." Until an international system of virus nomenclature is adopted, the authors prefer to use the name "ring spot virus."

History and Geographic Distribution

The wide distribution of the ring spot virus indicates that it probably has been present for a long time. One reason why its presence was not recognized earlier is its habit of chronic existence in most hosts without causing visible symptoms of the disease. The production of necrosis in various plant organs in the early stages of the disease has caused confusion with fungus and bacterial diseases, especially those of the leaf-spot and shot-hole types. Apparently the ring spot virus is actually a group virus composed of many forms, which because of their various effects on host plants have been reported in some cases as the causes of separate diseases.

Ring spot virus is prevalent in commercial peach orchards in western United States, where it exists in trees mostly without leaf symptoms and similarly in nursery stock propagated from them. The virus is also very commonly existent in almonds, prunes, plums, apricots, and cherries and has been recovered from native chokecherry, chickasaw plum, wildgoose plum, American plum, and escape plum seedlings. It is probably most widespread in cherries, being nearly universal in commercial orchards in western United States (*59, 156, 157*). The lesser occurrence in peach and other stone fruits in sections where cherries are not grown points to cherries as a primary reservoir (*58*). Moore and Keitt (*163*) reported that the necrotic ring spot virus is so prevalent in sour cherries in Wisconsin that they have been unable to find a source of sour cherry yellows virus free from it. Hildebrand reported ring spot on cherries (*84, 91, 95*) and on Italian Prune (*92*) in New York. Willison and Berkeley (*249*), in describing symptoms of tatter leaf of sweet cherry, and Willison, in describing a line pattern virosis of Shiro plum (*247*) and strains of the prune dwarf virus (*246*), described symptoms produced on peach and other hosts similar to those caused by ring spot.

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[18] Reeves, E. L. Unpublished data.

Economic Importance

The deleterious effect of ring spot is difficult to measure because the different forms of the virus have different effects, because damage on most hosts is limited to the acute stages of the disease, and because the horticultural varieties are affected differently. Symptoms are rare and obscure on most varieties of apricot, plum, and prune. Some forms of the virus diminish the crop on sweet and sour cherries during the initial stages of the disease and reduce the vigor of sweet cherries in the chronic state. Milbrath and Zeller (*156, 157*) showed that the virus produces dire and serious effects on oriental flowering cherries. Almonds are often strikingly mottled, but no data are available on reduction of crop yields. Damage to peaches varies with varieties, and with most forms of the virus it is limited to early stages of the disease. Varieties as severely injured as Rio Oso Gem when affected with some forms may be so severely killed back that the crop loss extends into succeeding years because of the loss of fruiting wood. Experimentally J. H. Hale, Early Hale, Rio Oso Gem, Rochester, and Fay Elberta have been shown to be permanently reduced in vigor when affected with severe symptom-producing forms of the virus.

Host Range

The virus has been recovered from many varieties of naturally infected peach (*Prunus persica*), sweet and sour cherries (*P. avium* and *P. cerasus*), Japanese plum (*P. salicina*), domestica plum (*P. domestica*), almond (*P. amygdalus*), and apricot (*P. armeniaca*). It has also been recovered from native stands of American plum (*P. americana*), chickasaw plum (*P. angustifolia*), wildgoose plum (*P. munsoniana*), sand cherry (*P. pumila*), eastern chokecherry (*P. virginiana*), and desert apricot (*P. fremontii*), from escape myrobalan plum (*P. cerasifera*) seedlings, from *Rosa* sp., and from Japanese apricot (*P. mume*). Experimentally ring spot virus has infected all the species of the genus *Prunus* on which it has been tried. The following species have been graft-inoculated and the virus has been recovered as evidenced by production of symptoms on peach after graft inoculation with affected material from the respective species: Peach, many varieties; mazzard cherry (*P. avium*) seedlings; mahaleb cherry (*P. mahaleb*) seedlings; chickasaw plum; myrobalan plum; almond; western chokecherry (*P. virginiana* var. *demissa*); domestica plum; Japanese plum; apricot; damson plum (*P. insititia*); Japanese apricot; desert apricot; flatwoods plum (*P. umbellata*); tangut almond (*P. tangutica*); David peach (*P. davidiana*); Bokar plum (*P. bokhariensis*); Manchu cherry (*P. tomentosa*); oriental flowering cherry (*P. serrulata*); and apple (*Malus sylvestris*).

Symptoms

The symptoms on stone fruits are variable and can be arranged in a gradient due to the differential resistance of host varieties and species and to the existence of and differential effects of forms of the virus (*57*). On some host varieties and species and with some virus forms symptoms are predominantly chlorosis; on others symptoms include stunting and necrosis of leaf, cambial, cortical, and bud tissues. In most hosts leaf symptoms, whether chlorotic or necrotic, include rings, spots, and patterns made up of rings and spots. The size of rings is usually inversely proportional to the number present. In all hosts symptoms are more pronounced in the early stages of the disease, and in many hosts visual leaf symptoms occur only during the first year of infection. Some hosts become infected and retain the virus without any visual symptoms. Some hosts develop leaf symptoms when infected with certain forms of the virus but not with others. In some hosts affected with certain forms of the virus visual symptoms occur annually; in such cases symptoms are usually more severe during the acute stages of the disease than during succeeding ones.

Peach.—Peach varieties differ in response to the ring spot virus. In one series of hybrid seedlings inoculated with a single form by Cochran some showed only a few obscure rings; others developed numerous rings, shot hole, bud killing, and tip dieback; and still others were entirely killed. Of the commercial varieties tested, Rio Oso Gem is one of the most severely injured (pl. 13, *A*). Affected trees in the acute stages are retarded; many buds, both flower and leaf, die when partially open; and past-season terminal growth may be killed and twig blight similar to that characteristic of brown rot, caused by *Sclerotinia* spp., may result. On some twigs not entirely killed, necrotic cankers form at the nodes and around the base of lateral buds. Leaves arising from buds partially killed show all grades of patterns from chlorosis to crowded ring patterns and shot hole (fig. 25).

On varieties having red-colored fruit, like Rio Oso Gem, J. H. Hale, and Rochester, the borders of rings and necrotic spots develop red margins (pl. 13, *C*). Initial leaves having necrotic rings or crowded ring patterns are usually shed during the first few weeks of growth (fig. 26). Trees severely killed back usually develop sucker shoots on the main trunks or rootstocks and appear to recover, and those affected with most forms of ring spot virus are devoid of leaf symptoms during the remainder of the season and in succeeding years (pl. 13, *B*). Fay Elberta peach trees inoculated by Cochran in series with forms varying in severity of symptom production were hedgerow-pruned 3 years after inoculation and observed for vigor. The trees which originally were severely affected made only approximately two-thirds the length of growth after pruning that the checks did (pl. 13, *F*), whereas those slightly affected equaled the checks in growth.

Fay Elberta, Late Elberta, Krummel, Rochester, and J. H. Hale are also severely affected. Nursery trees of these varieties inoculated with virus forms that had produced a gradient of symptoms were correspondingly affected. Some forms obtained from sweet cherry completely killed the inoculated trees. Others caused severe necrosis of buds, tip dieback (pl. 13, *E*), and killing of cambial and cortical tissues at the inoculation point, the last resulting in splitting and cracking of the bark (pl. 13, *D*) and often girdling of the entire arm. Trees inoculated with so-called mild forms are usually slightly retarded and pale in color. They develop scattered leaves with obscure spot and ring patterns, but they soon recover by shedding or greening of such leaves.

Some varieties such as Sims, Paloro, Tuskena (Tuscan), and Lovell are only mildly affected by forms which severely injure J. H. Hale. On these

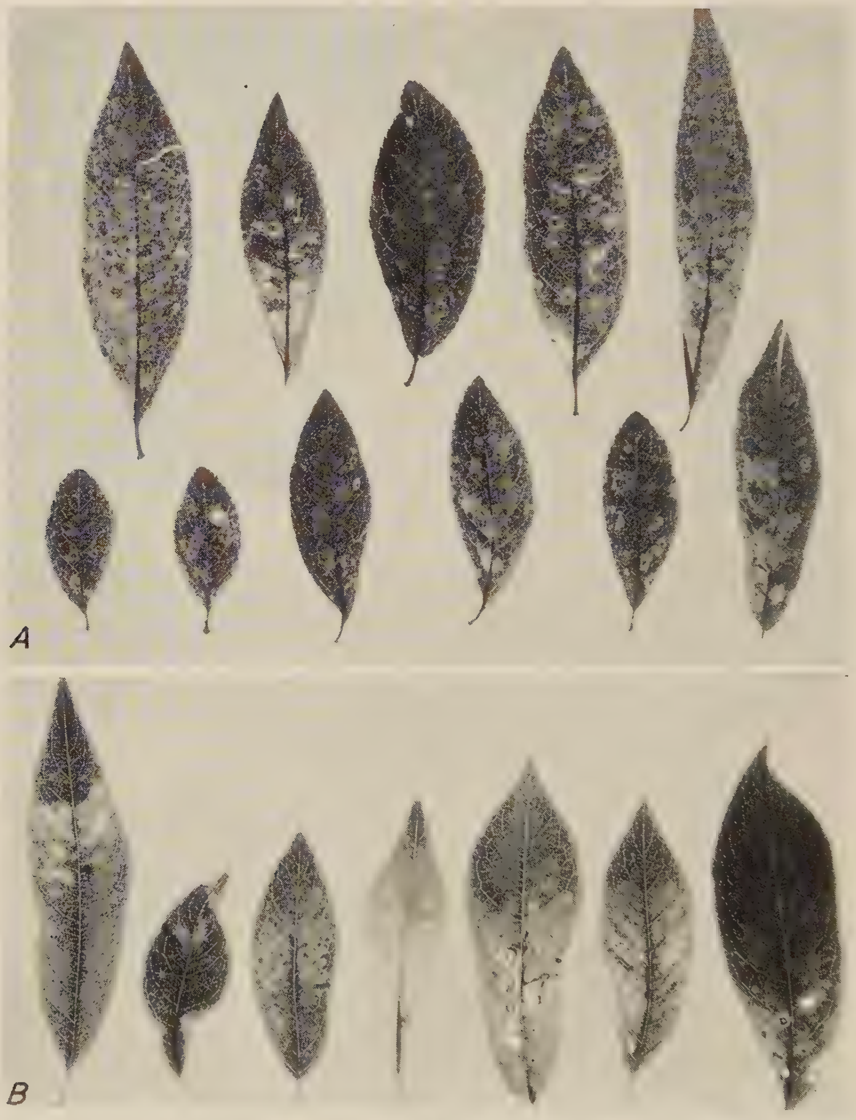

Figure 25.—*A*, Ring-spot-affected leaves of Lovell peach, showing rings, spots, and chlorotic patterns. *B*, Affected leaves of July Elberta peach on which the symptoms are predominantly chlorosis but on which rings and shot hole occasionally occur; leaves like third to fifth from the left are usually shed during the acute stages, and ones like the others become green as the season progresses.

varieties symptoms often consist of scattered ring and shot-hole patterns which may persist late into the growing season. Likewise, leaf symptoms produced by virus forms that cause mild effects on the more seriously damaged varieties may persist longer than those accompanying severe injury.

Plum.—On Japanese- and domestica-type plums symptoms of ring spot

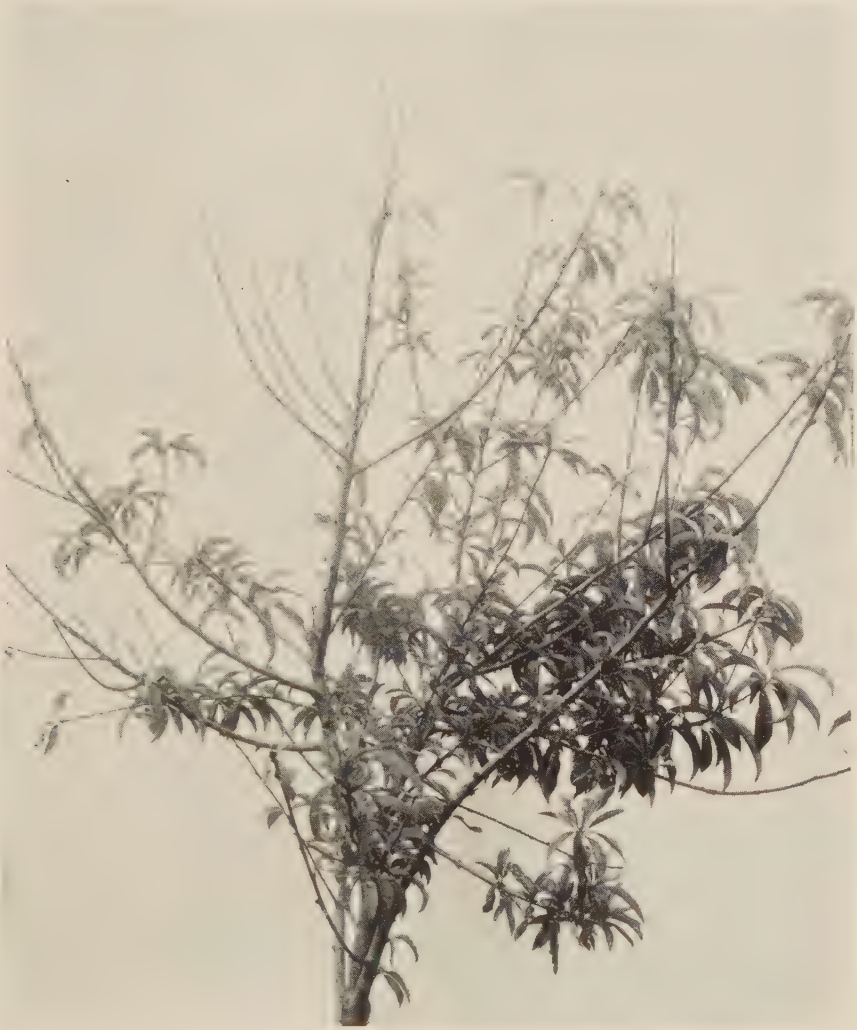

Figure 26.—**Elberta peach in midsummer after showing acute symptoms of ring spot in spring. The bare twigs and thin foliage show where the buds and twigs have died and been shed.**

are generally absent or mild. Some virus forms produce no symptoms on any varieties, and others produce them on only certain ones. Rings and chlorotic patterns were produced on French (Agen) and Hungarian prunes and on Shiro and Beauty plums. Myrobalan seedlings vary in reaction from no symptoms to chlorotic ring and fish-net patterns and in a few cases to necrotic spots and shot hole. No symptoms were seen on either bud-inoculated or naturally infected wildgoose and chickasaw plums. During acute stages of infection vivid chlorotic ring and mottle patterns were produced on Bokar and American plums.

Almond, apricot, and David peach.—On almond most forms of the virus cause symptoms that vary from chlorotic, crowded ring to oak-leaf and necrotic patterns (fig. 27, *B*). Expression is most striking in the Ne Plus Ultra, Nonpareil, I. X. L., Drake, and Peerless varieties in descending order. The disease is very prevalent in commercial orchards; and, while symptoms are always more evident the first year of the disease, they are often persistent in succeeding years in scattered leaves.

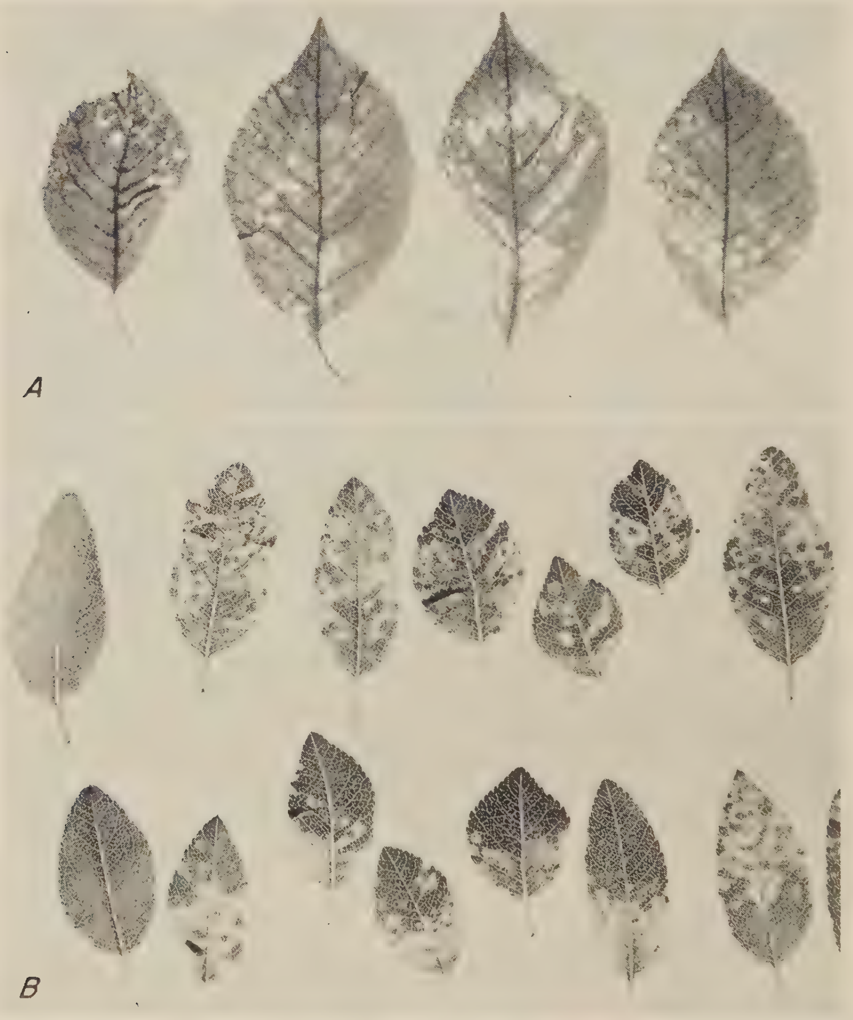

Figure 27.—*A*, Leaves of mazzard cherry affected with ring spot showing rings, shot hole, and chlorotic and necrotic patterns; *B*, affected Nonpareil almond leaves showing ring spot and chlorotic patterns in contrast with unaffected leaves at left.

Only a small proportion of the virus forms cause symptoms on apricot. These vary from sharply defined rings to oak-leaf patterns and necrosis. Occasionally, forms which cause tip blight and shot hole have been found.

On tangut almond and David peach the symptoms were similar to those on Bokar and American plums.

Cherry.—The ring spot virus is nearly universally present in commercial orchards of sweet and sour cherries in western United States, as evidenced by transmission to peach and oriental flowering cherries. Symptoms observed on sweet cherry in commercial orchards vary from chlorotic ring patterns to shot hole and lace leaf. On some trees symptoms persist annually, but are not as pronounced as first-year symptoms. Most sweet cherry trees from which transmission was effected were showing no symptoms when the inoculum was taken. Mazzard and mahaleb cherry seedlings inoculated with diseased peach tissue in the fall produced typical symptoms at the beginning of growth the following spring, varying from sharply defined rings and crowded ring patterns to shot hole and lace leaf (fig. 27, *A*). Individual seedlings, especially mahaleb, reacted variably when inoculated with material from a single source. Symptoms like those observed in the orchard have not been obtained

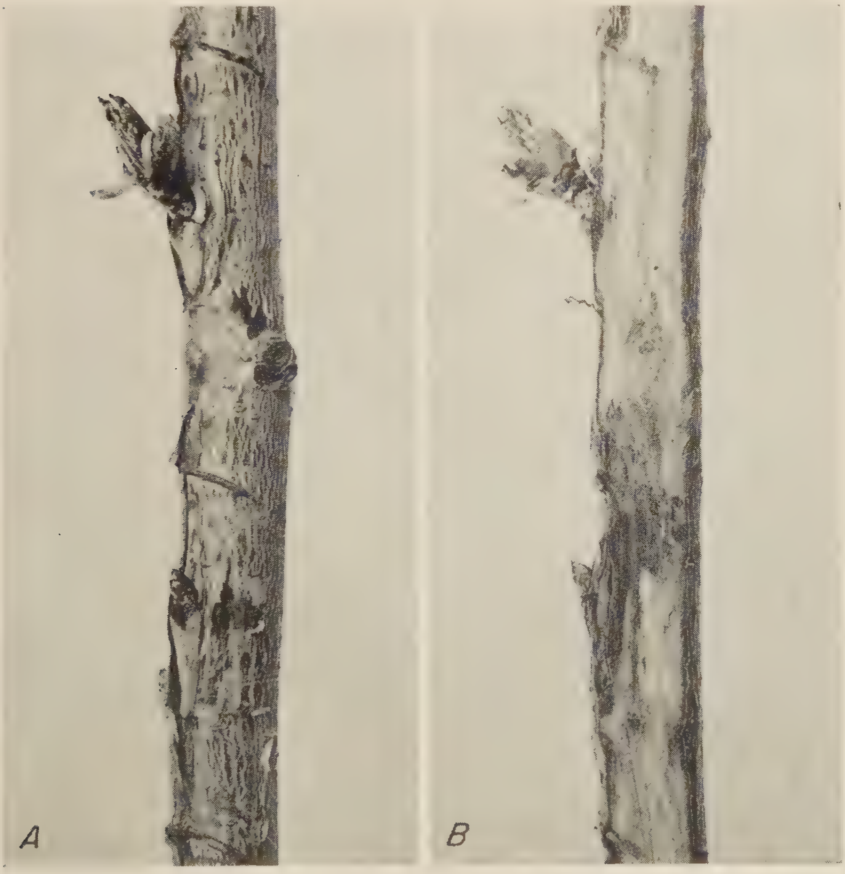

Figure 28.—A Shiro-fugen oriental flowering cherry stem budded with two sweet cherry buds (upper virus-free and lower infected with ring spot virus): *A*, Without bark removed; *B*, with bark removed to show necrosis of the tissues adjacent to the infected bud, the callus union, and the start of growth of the virus-free bud.

by experimental infection of sour cherry, since no virus-free sour cherry stock was available to the authors when these studies were made.

Milbrath and Zeller (*156, 157*) described the symptoms of ring spot and latent viruses on the Kwanzan and Shiro-fugen varieties of oriental flowering cherry. These varieties show promise as excellent diagnostic hosts in that they are seriously damaged by many if not all forms of the virus. On Shiro-fugen nearly all forms of the virus are apparently parenchyma-limited. When trees are inoculated with diseased buds the virus moves slowly from these diseased buds into the surrounding tissue. The tissue invaded becomes necrotic (fig. 28), and if left unimpeded the virus will eventually move down the stem, killing the whole plant. If the branch is removed to a point below any necrotic tissue, the progress of the disease is stopped; thus apparently the virus is removed. A single tree of the Shiro-fugen variety can be used to index a large number of orchard trees by spacing the inoculation buds and taking readings before girdling takes place. In Kwanzan the virus is more completely systemic (fig. 29). If buds from a tree under test show no reaction on Shiro-fugen they are tested on Kwanzan. Milbrath and Zeller found a few forms which react on Kwanzan but not on Shiro-fugen and conversely.

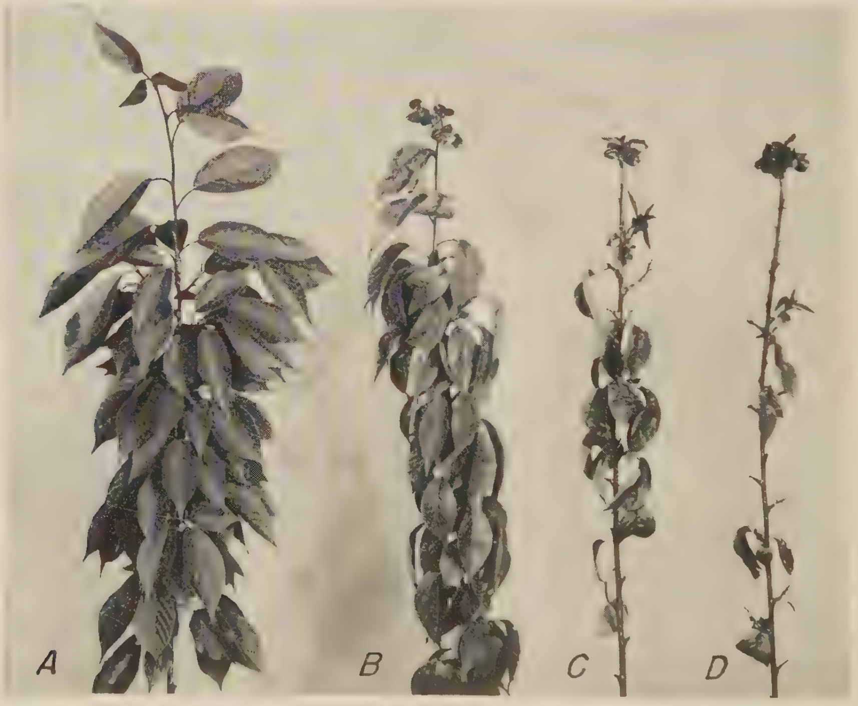

Figure 29.—*A*, Branch from normal Kwanzan oriental flowering cherry tree; *B–D*, branches from comparable trees inoculated with three forms of the ring spot virus producing a gradient of symptoms: *B*, Mild; *C*, intermediate; *D*, severe.

Transmission

Transmission has been experimentally effected only through tissue grafting. Cochran (*58, 59a*) showed that the virus passes through a low percentage of the seeds of mazzard cherry and of Lovell and Rio Oso Gem peaches; ring and mottle patterns were produced on the mazzard seedlings, but no symptoms were seen on the peach seedlings. Cation (*51*) found upward of 10 percent of mahaleb cherry seedlings grown from commercial seed carrying the ring spot virus. The infected mahaleb seedlings showed no symptoms, and the presence of the virus was demonstrated by indexing on peach. Spread recorded by Cochran and Stout in commercial peach and almond orchards varied in rate in different years.

When peach buds containing the ring spot virus are budded on healthy Lovell peach seedlings or trees of various peach varieties, the buds commonly die without making any perceptible union, yet a high percentage of the seedlings or trees become infected. Conversely, when healthy buds of a variety are placed on a stock containing the virus, they likewise often fail to live. When buds containing the virus are placed on stocks also containing the virus there are no shock symptoms and no abnormal number of bud failures. Bud failure when the bud is carrying the virus and the stock is healthy is somewhat variable, depending largely on two factors: the relative severity of effect of the form of the virus present as expressed on severely damaged varieties and the relative susceptibility of the seedling or variety understock to injury.

A larger percentage of peach buds containing severe-symptom-producing forms of the virus will fail on all peach varieties than of buds containing only mild ones. Correspondingly, even buds containing only so-called mild forms may fail on peach varieties which are strong reactors. Milbrath and Zeller (*156*) showed that healthy buds of oriental flowering cherry fail when they are placed on mazzard seedlings or cherry varieties containing the ring spot virus. No appreciable difference was noted in the number of failures of apricot, almond, and plum buds carrying ring spot virus and of those free from it when they were placed on Lovell peach. The failure of buds is believed to be due to the killing of newly invaded cells of the stock adjacent to the diseased bud shield, which prevents union of shield and stock.

Incubation Period

The incubation period varies with the condition of the tree inoculated. Peach trees inoculated while breaking dormancy sometimes develop symptoms in new leaves at terminals in as short a time as 10 days. Trees just reaching full leaf usually require up to 3 or 4 weeks, and the resulting infection is usually limited to a portion of the tree. Trees inoculated as late as June to August may not show symptoms in leaves until the following growing season; but, when infected with forms of the virus which cause canker and bark necrosis, they may develop sunken bark cankers adjacent to and usually below the inoculation point during the current season. Trees inoculated in the fall just before the bark becomes tight develop symptoms on the first growth of the following season. The virus commonly invades all parts of small nursery trees during the first season, but symptoms on large trees are often limited to the inoculated arm and the basal portion of adjacent arms.

Control Measures

The almost universal latent occurrence of the ring spot virus, the evidence of its rapid tree-to-tree spread in many stone-fruit orchards, and the lack of evidence that the virus is associated with serious general reductions in yield make the feasibility of control by ordinary methods questionable. Some workers feel that reduction in crop during the acute stages of the disease and the possible reduction in vigor of trees in the chronic stages are worthy of efforts to furnish foundation stock free of the virus for the nursery trade.

Remarks

The widespread occurrence of the ring spot virus in many hosts, especially in the Pacific Northwest, is evidence that it has been present for a long time. On peach, ring spot has with little doubt been confused with the twig blight symptom of brown rot, caused by *Sclerotinia* spp., and the twig blight stage of peach blight, caused by *Coryneum carpophilum* (Lév.) Jauch. The latent character of ring spot in the chronic state in many hosts has allowed the causal virus to be carried as a contaminant with other viruses in tissue transmission and has resulted in confusion, especially in host-range studies. If two viruses exist in a single host, only one of which has effected symptoms, and both are transferred in tissue to a second host, there is no basis for ascertaining which is responsible for the symptoms in the second (*59*).

The relation of ring spot and necrotic ring spot of sour cherries needs more study to ascertain whether they are identical. The virus causing ring spot as expressed on peach is universally present in sour cherry on the west coast, and no healthy sour cherry material was available during early studies for inoculation studies on sour cherry. Sour cherries carrying ring spot virus without symptoms develop no symptoms after inoculation with peach buds carrying the virus. Hildebrand (*84, 91*), Rasmussen and Cation (*176*), Keitt and Clayton (*124*), Berkeley and Willison (*9*), and Willison, Berkeley, and Chamberlain (*250*), working with sour cherry yellows, described symptoms on peach and other hosts similar to those of ring spot; this suggests that the ring spot virus was present in their cultures or is a component of the cause of sour cherry yellows. Moore and Keitt (*163, 164*) showed that necrotic ring spot virus is a single entity but were not able to obtain a culture of sour cherry yellows free from it. It seems logical, on the basis of wide occurrence, variation in expression of ring spot, and the interhost-transmission results obtained by various workers, that necrotic ring spot of sour cherry, tatter leaf of sweet cherry, and possibly others, already described, belong to the ring spot group.

PEACH NECROTIC LEAF SPOT

By Donald Cation

Name of the Disease

This disease, originally described on peach (*49*), was called necrotic spot to distinguish it from ring spot (*62*).

Name of the Causal Virus

No Latin binomial has been applied to the causal virus, but for convenience it may be referred to as necrotic leaf spot virus.

History and Geographic Distribution

Necrotic leaf spot was first observed in 1940 on two Elberta and two Carman peach trees which had been inoculated in the fall of 1939 during a routine indexing test with buds from a Windsor sweet cherry tree located in East Lansing, Mich. Further inoculations from these peach and Windsor cherry trees to additional seedlings and varieties of peach consistently resulted in the same pattern of symptoms, which were different from the ring spot symptoms of the types described by Cochran and Hutchins (*62*), Berkeley (*8*), Willison and Berkeley (*249*), and Hildebrand (*91*). Additional indexings from sweet cherries in Michigan indicate that the virus of necrotic leaf spot is frequently present as a latent virus in sweet cherry and as a result of distribution by nurseries it may be widespread in that species. The virus is frequently found in sweet cherries in California as indicated by indexing on peach.[19]

Economic Importance

The harmful effects on peach and cherry have not been measured, but the disease appears to be of small consequence.

Host Range

The disease has been transmitted to seedlings and seven varieties of peach (*Prunus persica*), namely, Elberta, Carman, J. H. Hale, Golden Jubilee, South Haven, Halehaven, and Fertile Hale, and causes symptoms on them. No symptoms have been observed on the varieties of sweet cherry (*P. avium*) and sour cherry (*P. cerasus*) tested, but the sweet cherry varieties tested were found to be symptomless carriers.

Symptoms

Peach.—In the orchard, light-brown, membranous, dead areas appear only on young, unfolding leaves of affected peach trees (fig. 30). The largest numbers of characteristic necrotic spots occur on unfolding leaves during July, but a few typical spots may be found earlier. Traces of necrotic spotting are occasionally observed in the spring on the first-formed leaves. The necrotic spots are usually round and may occur on any part of the leaf blade. Occasionally a long, narrow area along the leaf margin is affected. Rarely more than one or two leaves on any particular growing shoot are affected. The dead tissue soon falls out, leaving a clean-edged margin. Other parts of the leaves are of normal color. Affected leaves do not drop prematurely. Occasionally faint chlorotic spots accompanied by leaf distortion are seen

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[19] Conversation with L. C. Cochran at Beltsville, Md., August 26, 1949.

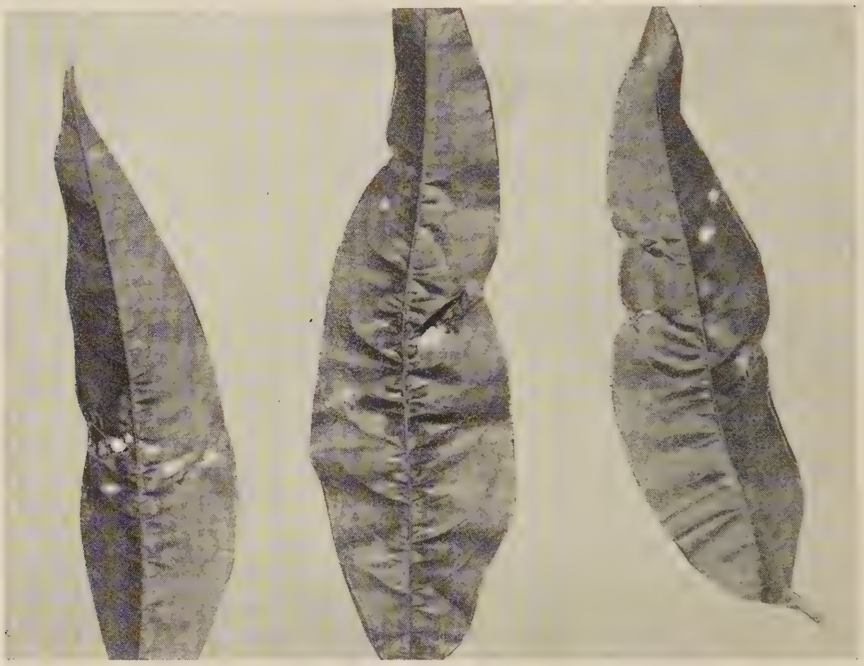

Figure 30.—**Young leaves from peach trees affected with necrotic leaf spot.**

early in the season, but these symptoms occur on relatively few leaves. Inoculations by budding of peach trees in August do not result in delayed foliation, dieback, or bark necrosis in the following spring, although such symptoms are common on peach inoculated with certain strains of ring spot virus (*8, 62, 91*).

In 1947 seedling peach trees in the nursery row inoculated with necrotic leaf spot virus did not show delayed foliation and dieback of the previous year's growth as did those inoculated with the strains of ring spot virus, but they did show dieback of one or more shoots of the current year's growth 2 to 3 weeks after bud break (May 23). In June and July typical necrotic spot developed on some of the leaves on other shoots.

Inoculation of Fertile Hale peach trees with buds of Windsor cherry containing the necrotic leaf spot virus at the time of growth initiation resulted in symptoms not unlike those resulting from strains of ring spot virus. The symptoms on peach in the greenhouse consisted of dieback of some of the new shoots and chlorotic and necrotic ring spots on leaves. The inoculum may have been contaminated with strains of the ring spot virus (*59*).

Cherry.—Certain symptomless Windsor, Black Tartarian, and Napoleon (Royal Ann) sweet cherry trees were shown to be carriers of the virus by indexing on peach. Inoculations of the same varieties of cherry did not result in symptoms. Inoculations of Montmorency sour cherry trees resulted in no symptoms, but trees from the same lot were later shown to be carrying ring spot virus similar to that reported by Berkeley (*8*) and Hildebrand (*91*).

Diagnostic or Unusual Characteristics

Necrotic leaf spot is distinguished by the necrotic areas on new leaves unfolding in midseason and by the absence of chlorotic rings, bark necrosis, dieback of previous season's growth, and delayed foliation which generally accompany ring spot diseases in peach. The chronic nature of the disease as characterized by the appearance of symptoms year after year and in midsummer is also at variance with the ring spot diseases reported (*8, 62, 91, 249*).

Transmission

Transmission as far as known is effected only by budding or other forms of the grafting process. Indications of natural spread have not been observed.

Incubation Period

Inoculations in August in the field result in symptoms the following season, 9 to 10 months later, but symptoms are obtained in 3 to 5 weeks when trees just breaking dormancy are inoculated in the greenhouse.

Control Measures

The disease can be avoided in the propagation of sweet cherry by selecting scion wood from trees previously determined to be disease-free by indexing on peach. Peach trees affected with necrotic leaf spot should not be used as a source of propagating material.

ASTEROID SPOT

By L. C. Cochran and C. O. Smith

Names of the Disease

The name "asteroid spot" was given to this disease because of the similarity of the leaf spot symptoms on peach to small, star-shaped splotches (*65*). The disease is known locally in Texas as stipple spot, but it has no relation to the noninfectious condition on myrobalan plum seedlings described by Hildebrand (*94*) under the name "asteroid spot."

Names of the Causal Virus

Marmor astri Holmes is the only Latin binomial which has been assigned to this virus. It is referred to here by adding the term "virus" to the common name of the disease; thus, "asteroid spot virus."

History and Geographic Distribution

Asteroid spot was first seen on the peach understock of some trees in a rootstock experiment at the Citrus Experiment Station, Riverside, Calif. The occurrence in commercial orchards in California was first noted in 1939 and called to the authors' attention by R. L. McClain, who suggested its identity with a disease widely existent in Texas and temporarily designated by Lee M. Hutchins as "stipple spot." Further studies of asteroid spot showed variation in symptom expression due to the presence of virus forms, and it seems likely that stipple spot, as well as others, is the same as asteroid spot. Asteroid spot is widespread in peach in Texas and westward to southern California. Two affected trees reported in North Carolina were traced directly to infected nursery stock originating in Texas (*55*). Occasional affected trees have been seen in Oregon, Utah, and Colorado. One case was seen in a very old peach tree, probably a Spanish seedling, growing in the courtyard of the Convent de Monte Cristo, Puebla, Mexico.

Economic Importance

There is very little evidence that asteroid spot is the cause of any appreciable damage on any of the hosts. It may exist in certain hosts without causing symptoms, and it must thus be regarded in the light of a possible contaminant in host-range studies.

Host Range

Symptoms of asteroid spot have been seen on peach (*Prunus persica*), nectarine (*P. persica* var. *nectarina*), almond (*P. amygdalus*), apricot (*P. armeniaca*), Japanese plum (*P. salicina*), domestica plum (*P. domestica*), Japanese apricot (*P. mume*), and ansu apricot (*P. armeniaca* var. *ansu*). Peach, apricot, Japanese plum, and almond have been experimentally infected.

Symptoms

On peach symptoms first show about 6 weeks after growth starts, but they become most pronounced about August. Spots begin as small, translucent light-green flecks in the darker green of fully expanded leaves, giving the appearance of yellowish-green paint on the leaves (fig. 31). As the leaves mature, scattered leaves turn yellow but the fully formed spots remain yellowish green, thus reversing the color contrast. Spots vary in size, the size being inversely proportional to the number present. Occasionally some of the

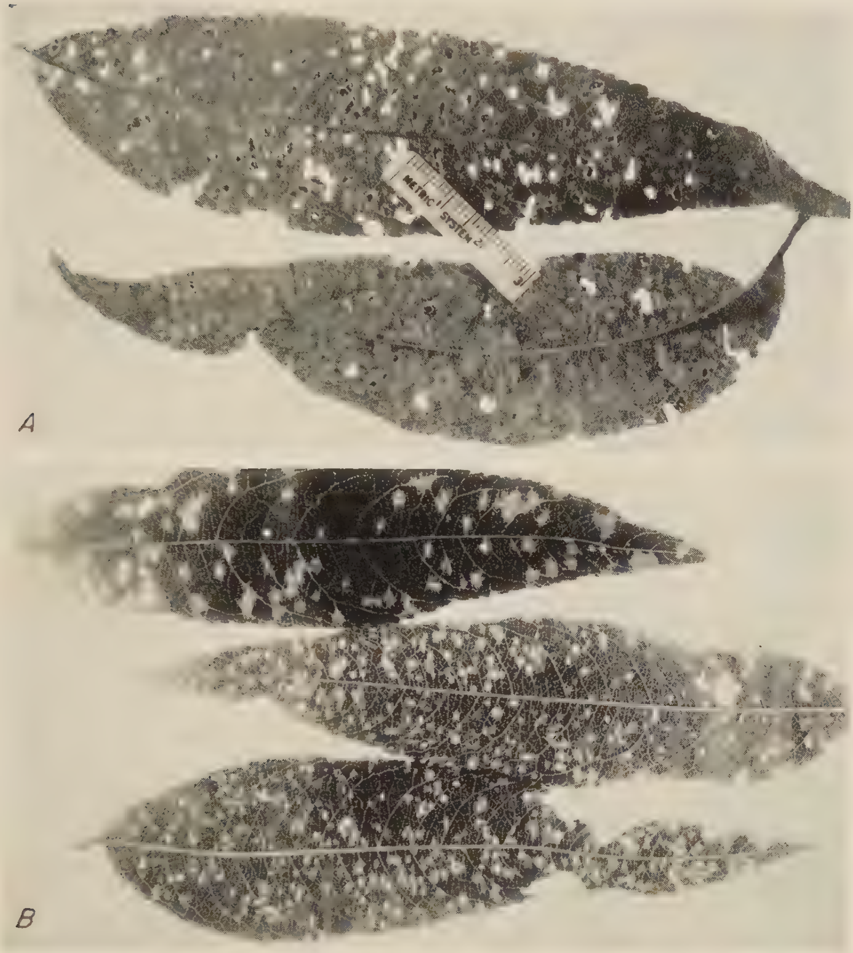

Figure 31.—**Peach leaves affected with asteroid spot: *A*, J. H. Hale, showing typical spots and occasional necrotic ones; *B*, Lovell.**

larger spots have green centers and thus are really rings. On some leaves large, angular, chlorotic blotches form along the veins as if the veins were ruptured and had flooded the bordering tissue.

In field comparisons white-fleshed peach varieties appear to develop more pronounced symptoms than yellow-fleshed ones. Similarly nectarines, which are white-fleshed, are strongly affected. On orchard trees symptoms are usually limited to chlorotic spots, but necrosis has been seen on inoculated J. H. Hale peach trees during acute stages. Symptoms are more pronounced during the initial year of the disease, but affected trees show symptoms annually. The size, intensity, and number of spots in inoculated peach trees varied with the sources of the virus, indicating the existence of forms of the virus.

On Japanese-type plum symptoms vary with varieties, but resemble closely those on peach. Late Santa Rosa (fig. 32, *A*) was strikingly affected, whereas regular Santa Rosa, Wickson, and Satsuma plums were less affected. On inoculated almond and French (Agen) prune the symptoms were obscure. On apricot (fig. 32, *B*) symptoms were well developed during the acute stages, but they failed to reappear after the trees became thoroughly affected. Transmission from naturally affected plums and peaches to apricot gave variable

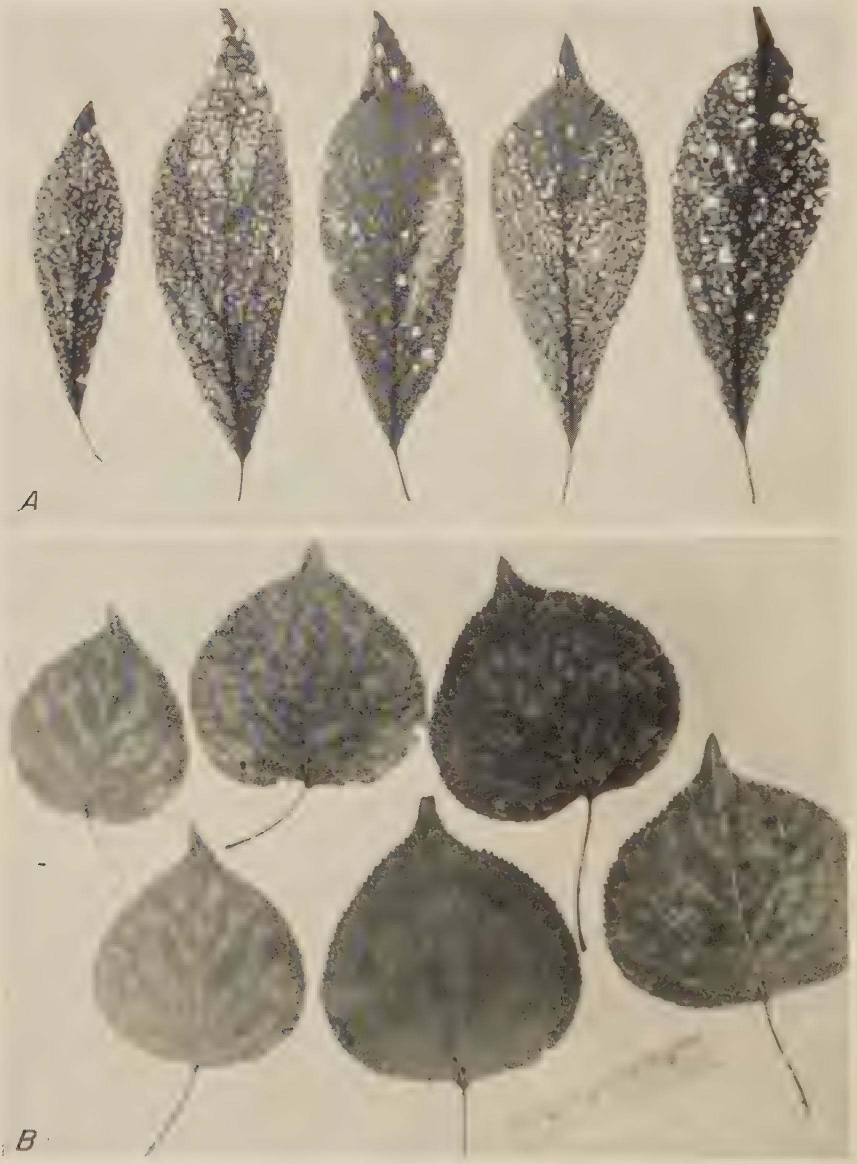

Figure 32.—Leaves of *(A)* Late Santa Rosa plum and *(B)* Royal apricot affected with asteroid spot, except for normal leaf at center of bottom row.

results, inoculum from some producing well-defined symptoms and that from others obscure symptoms or none.

Transmission and Incubation Period

Transmission from peach to peach by inoculation with bark shields in the fall usually results in symptom expression in fully expanded leaves 6 to 8 weeks after the beginning of spring growth. Transmission was effected experimentally in 8 weeks when infected scions were grafted on peach trees which were breaking dormancy. Translocation of the virus appears to be slow, and initial symptoms may be obscure but may become more intense as more tissue is involved. The widespread occurrence of the disease in some orchards is indicative of natural spread, but, if this takes place, the vector is unknown.

Control Measures

Available data do not show that the asteroid spot causes crop damage of any consequence, but it is reasonable that it should be avoided in the establishment of nursery foundation stocks.

Remarks

Asteroid spot is chiefly important because of the confusion that may result in interpretation of experimental work when the causal virus is carried in the cultures as a contaminant.

GOLDEN-NET

By E. W. Bodine and L. W. Durrell

Other Common Names of the Disease

None.

Name of the Causal Virus

Golden-net virus.

History and Geographic Distribution

Golden-net was found first in Colorado in 1937 on apricot and plum and in 1939 on Elberta peach (*36, 40*). It has also been observed in California.[20]

Economic Importance

Golden-net virus appears to cause little damage to peach and plum. It does, however, cause severe damage to apricot. To date, however, little natural spread of the golden-net virus has been observed in apricots.

Host Range

As far as known golden-net has been found only on Elberta peach (*Prunus persica*) and its seedlings, on an unidentified variety of apricot (*P. armeniaca*), and on Satsuma plum (*P. salicina*).

Symptoms

Peach.—The characteristic symptom on peach trees is the marginal yellowing of the veins of the affected leaves (fig. 33). Because of this outstanding constant symptom the disease is called golden-net. Occasionally irregular primuline yellow areas occur on some leaves. No retardation of twig growth is apparent, and fruits on affected trees are normal in size, shape, and color.

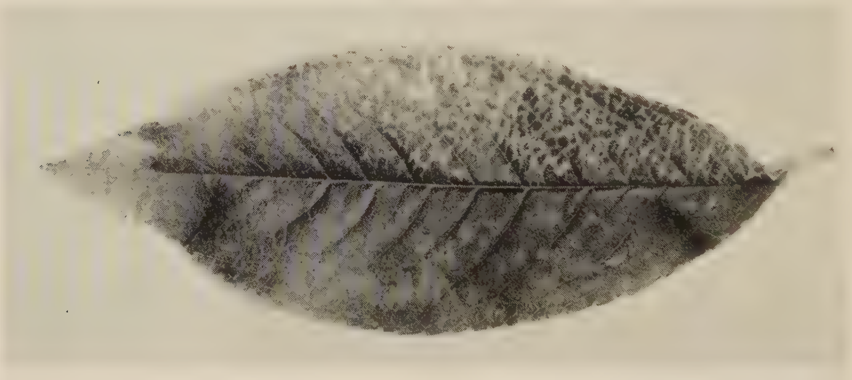

Figure 33.—Elberta peach leaf affected with golden-net.

Apricot.—The disease affects the apricot tree more severely than it does the peach. The leaves frequently show an abnormal crinkling and mottling along and between the veins. The current-shoot growth of diseased trees is distinctly stunted. After the stone-hardening period, the fruits become misshapen and exhibit a marked bumpiness.

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[20] Thomas, H. Earl. Unpublished data.

Plum.—The only symptom on Satsuma plum consists of an inconspicuous marginal mottle, frequently associated with irregular yellow areas on some of the leaves on infected trees in the early spring.

Transmission

Transmission has been accomplished only through budding.

Incubation Period

The incubation period after the insertion of diseased buds varied from 9 to 18 months.

Control Measures

It is not felt at present that this disease justifies control by diseased-tree removal, because of the small amount of damage on affected peach and plum and the lack of evidence of natural spread among apricots.

PEACH CALICO

By Earle C. Blodgett and E. L. Reeves

Name of the Disease

"Leaf variegation" is sometimes used for peach calico, but more properly this name refers to the nontransmissible, or chimera, type of disease.

Name of the Causal Virus

Peach calico virus.

Geographic Distribution

One naturally diseased tree (*19*) and two other trees possibly diseased have been seen in Canyon County, Idaho. Nursery trees were experimentally infected in Latah County. Peach calico has also been reported in Washington (*184*). Two diseased trees have been observed in King County and four in Chelan and Douglas Counties.

Economic Importance

Peach calico at present is regarded as of very minor importance. Losses might result, however, if nursery stock was propagated from an infected tree, since a goodly portion of the stock might show symptoms and be unsalable. The total effect of the disease on a tree is minor after the initial stages.

Host Range and Varietal Susceptibility

Rochester, Early Crawford, and Elberta varieties of peach (*Prunus persica*) were found naturally affected. Transmission to the Elberta variety and to peach seedlings has been effected with buds. Three J. H. Hale peach nursery trees failed to show symptoms when inoculated. Thomas and Rawlins (*233*) reported that almond calico is transmissible to peach and cherry, but Thomas stated that he does not believe peach calico found in Idaho is the same as calico on almond.[21]

Bing (*P. avium*), Montmorency (*P. cerasus*), and May Duke (hybrid) cherries, Moorpark apricot (*P. armeniaca*), Italian Prune (*P. domestica*), and Ne Plus Ultra almond (*P. amygdalus*) were inoculated in limited trials, but no symptoms developed on any of these. When the inoculation buds grew into shoots, leaves on them showed typical symptoms. No data are available to indicate whether the varieties listed are symptomless carriers.

Symptoms

Symptoms of peach calico may appear as the leaves unfold, showing in the early stages a mottling, or mosaiclike, pattern. Later the light-green areas enlarge, principally along the veins, and eventually the color changes to a brilliant yellow or almost papery white (fig. 34). Not all leaves show symptoms. Creamy-white streaks or nonuniform patterns develop on affected twigs. The margins of these streaks or areas may be pink. The difference between calico and leaf variegation (nonvirus) is fairly distinct. In leaf variegation the affected areas are rather sharply delimited with at least three distinct shades of green, while in calico the veins are yellow and there is a gradient of color in leaf tissue from green to yellow or white (*24*).

Affected trees usually show fewer and less distinct symptoms the second or

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[21] Thomas, H. Earl. Correspondence.

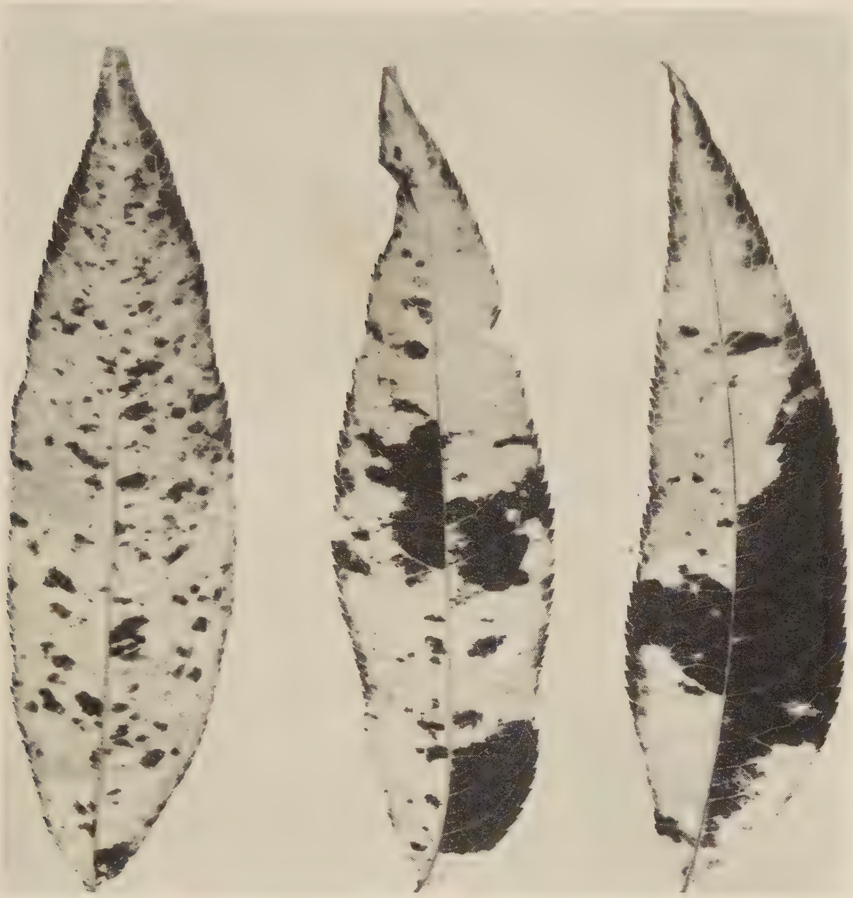

Figure 34.—**Peach leaves affected with calico.**

third year of the disease and may finally even become symptomless. Fruits on affected branches may show symptoms when they are about the size of walnuts. The peaches are smaller, shorter, and more nearly round than normal ones. There may be irregular patches of creamy-white tissue showing through the pubescence. In a few cases the affected areas are orange, shading into dull red.

Diagnostic or Unusual Characteristics

Careful examination of leaf symptoms and comparison with symptoms of leaf variegation are essential in diagnosis. Of particular interest is the evidence, both on the original tree and on experimental stock, of erratic, unpredictable behavior of the peach calico virus.

Reeves found that 10 of 11 Elberta peach trees receiving inoculating buds from an affected Early Crawford tree exhibited leaf symptoms of peach calico the first year after inoculation; only 1 of the 10 trees showed leaf symptoms the second year after inoculation and these were meager; and none exhibited symptoms the third year. Similar results were obtained with buds taken from an affected Rochester tree budded into healthy Elberta trees. Buds taken from 1 previously inoculated Elberta tree the second year after infection (a tree which had exhibited symptoms of calico the first year but not the second) resulted in the reproduction of symptoms on 1 of 10 trees receiving such inoculating buds. This preliminary test might indicate that the virus could be latent but of limited distribution in a peach tree not exhibiting foliage symptoms, but further tests should be made to establish these points.

Transmission

Positive transmission has been obtained by bud inoculation from peach to peach, but many attempts were unsuccessful. It appears that movement of the virus in a tree is slow and that the extent of distribution is uncertain.

Incubation Period

Trees budded in the fall with calico-infected buds exhibit symptoms on the scion early in the spring, but visible infection of the leaves of the stock may not appear until past midsummer.

Control Measures

Infected trees should be removed, and budwood from healthy trees should be used for propagation purposes.

Remarks

Observational and experimental evidence indicate that at present peach calico is of more academic interest than economic importance. Further study is desirable because of its unusual characteristics and erratic behavior.

PEACH BLOTCH

By R. S. Willison

Other Common Names of the Disease

None.

Name of the Causal Virus

Peach blotch virus.

Geographic Distribution

Peach blotch has been found on single trees in two widely separated orchards on Niagara Peninsula, Ontario, Canada.

Economic Importance

Peach blotch is of minor importance.

Host Range

So far the only species known to be attacked by blotch is peach (*Prunus persica*). The disease has been found on an unidentified variety and on Marigold in nature, and it has been transmitted experimentally to the varieties Elberta, Rochester, Vedette, Golden Jubilee, and Peregrine.

To date, transmission experiments with plum, apricot, and cherry varieties and cherry seedlings have given negative results except for a vague and doubtful blotching on Italian Prune. The species and varieties used in these experiments included domestica plum (*P. domestica*) varieties Italian Prune, German Prune, Reine Claude, and Lombard; Japanese plum (*P. salicina*) variety Abundance; myrobalan plum (*P. cerasifera*) seedlings; apricot (*P. armeniaca*) variety Niagara; sweet cherry (*P. avium*) varieties Black Tartarian and Napoleon (Royal Ann) and seedlings; sour cherry (*P. cerasus*) variety Montmorency; and mahaleb cherry (*P. mahaleb*) seedlings. Whether or not plums and cherries can act as carriers of blotch has not yet been determined.

Symptoms

The symptoms of peach blotch are well-defined pale-green to yellow-green variegations of the leaves (fig. 35). Up to the present no symptoms have been observed on flowers, fruits, or twigs. The variegations may be numerous, small, angular spots scattered over the leaf surface or less numerous, large, irregular blotches. Sometimes there is a single chlorotic area arranged symmetrically along the midrib and having its margins either well defined or feathered out along the lateral veins. Some leaves are marked only by a yellowing of the veins, and others may be symptomless. The patterns may also be reversed, dark-green areas on a chlorotic background. The margins of some leaves become scorched and soon drop off, leaving a ragged edge, or less frequently large necrotic spots may develop. On some varieties, such as Elberta, and on some seedlings, most of the pattern types can be found (*248*). The feathery-edge pattern predominates on Rochester leaves, while leaves of Vedette show a few large blotches appearing in some seasons but not in others. Golden Jubilee is usually symptomless, though in some years a few early leaves may have a mild mosaic or a yellowing of veins. Some seedlings exhibit either faint symptoms or none.

Diagnostic or Unusual Characteristics

Peach blotch is unusual in its isolated occurrence in nature. It differs

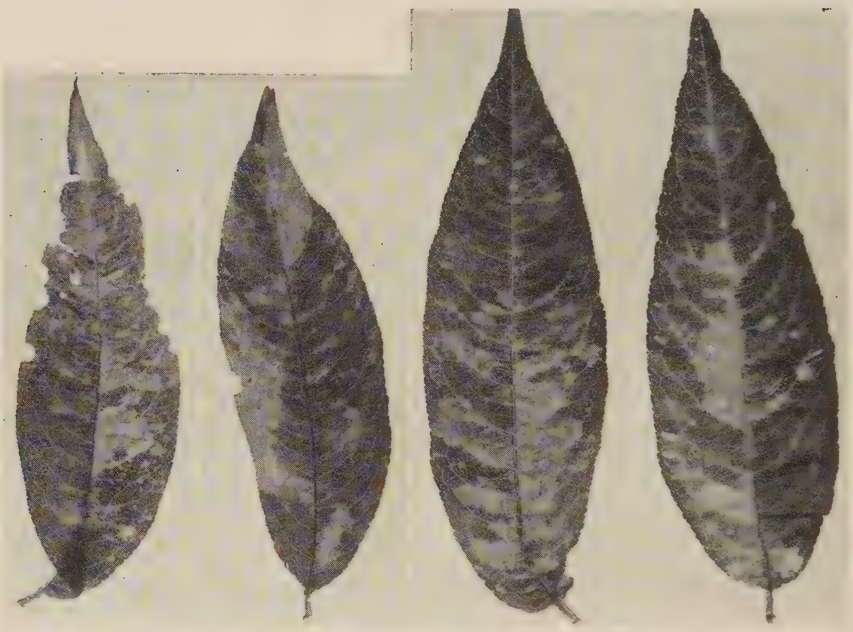

Figure 35.—Peach leaves affected with peach blotch.

from peach mottle in that it causes no symptoms in cherry and from peach calico (*24*) in that there is an absence of symptoms on twigs and fruits.

Transmission

Transmission has been effected only by budding and grafting. There is as yet no evidence that this disease spreads in the orchard.

Incubation Period

Symptoms appear on trees in the spring after late-summer inoculation.

Control Measures

Selection of disease-free scion and stock material for propagation is recommended.

Remarks

It is still an open question whether diseases like peach blotch are the result of sporadic infections involving preexistent viruses or originate within the host as a result of alteration in some part of the host's cellular components.

WILLOW TWIG

By H. Earl Thomas

Name of the Disease

The death and shedding of a majority of the lateral leaf buds on peach, resulting in slender, pendulant twigs similar to those of certain willow species, suggest the name "willow twig." A rough bark condition on some branches of affected trees suggests that this disease may belong to the rough bark group.

Name of the Causal Virus

No Latin binomial has been applied to the causal virus.

Geographic Distribution

Willow twig is fairly common from Sutter County south to Merced in central California and occurs less frequently in certain of the coastal counties (*231*).

Economic Importance

Affected trees may live indefinitely, but their fruits are inferior in quality and quantity, perhaps because of the reduced amount of foliage. The disease is the cause of local losses. The chief threat is that it may be spread in nursery stock by undiscerning propagators.

Host Range

The disease has been found to affect peach (*Prunus persica*) varieties J. H. Hale, Phillips, and Paloro, flowering peach, and nectarine (*P. persica* var. *nectarina*) variety Quetta.

Symptoms

The principal feature of this disease is the dying and shedding of lateral leaf and fruit buds during the winter and the resultant willowy growth and sparse foliage (fig. 36). On J. H. Hale peach, fruit buds are more persistent than leaf buds, but if leaf buds are lost, the fruits on these spurs do not mature properly. When a lateral bud grows as much as half an inch during the season in which it is formed, it is likely to survive and produce a side branch the following year. The main terminal bud of a branch usually survives but occasionally does not; in such a case progressive dieback results in loss of the whole branch. Affected branches at 5 years of age may be no thicker than a normal 1-year-old branch and are more flexible than a normal branch of the same diameter. On affected trees the first leaves in spring tend to be shorter in proportion to width than is normal for the variety.

On affected branches of Paloro, leaves tend to persist longer than on normal ones, often to mid-December, and some have a yellow band around the edge extending halfway to the midrib. Symptoms vary in severity on different branches on the same tree.

Symptoms appear to be more pronounced on trees in the warmer parts of the interior valleys. The effect of temperature is further supported by production of more pronounced symptoms on trees in the greenhouse than on trees growing out of doors at Berkeley. Heavy applications of nitrogen have produced little effect on the symptoms picture. Spring pruning by cutting off terminals of the current-season growth has not been effective in reducing the symptoms on new growth.

Transmission and Incubation Period

Willow twig has been transmitted by grafting diseased scions onto nursery-size trees, but three or more years are necessary for characteristic symptoms to develop. The rate of natural spread appears to be slow.

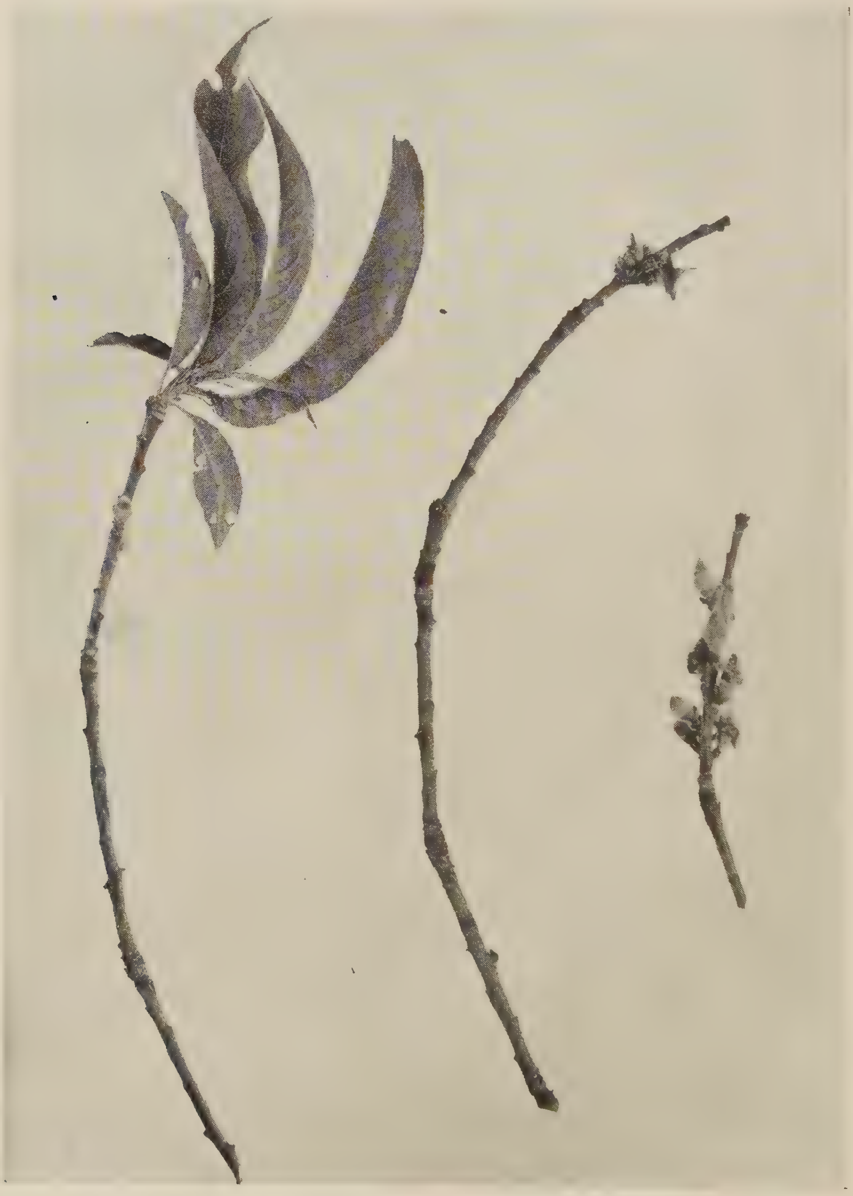

Figure 36.—Twigs of J. H. Hale peach affected with willow twig.

Control Measures

The most effective control measure is the use of healthy budwood sources for propagation of nursery stock. Although the disease spreads slowly in the orchard, there is little likelihood that it can be eradicated by diseased-tree removal. The causal virus may be present in a tree for three or more years before recognizable symptoms develop. Early-stage symptoms are not clear-cut and may be confused with other conditions. The effects of insufficient chilling are very similar to the symptoms of willow twig.

Remarks

The symptoms of willow twig on peach are similar in general to those of the type of bud failure disease of almond that is reported to be nontransmissible. Insufficient work has been done to determine the possibility of any relationship between these two diseases.

VIRUS DISEASES OF SWEET CHERRY

BUCKSKIN

By T. E. Rawlins and H. Earl Thomas

Names of the Disease

"Buckskin" is the name used for a destructive graft-transmissible virus disease of sweet cherry in California (*178*). On peach, however, the disease was called leaf-casting yellows (*234*) before it was proved that it was caused by the buckskin virus. X-disease (*226*), western X-disease (*265, 265a*), and yellow-red disease (*103*) have been used to designate a disease of peach and chokecherry apparently caused by various strains of the buckskin virus. In Oregon a disease of sweet cherry called albino cherry may be related to buckskin (*266*).

Name of the Causal Virus

Buckskin virus.

Geographic Distribution

Buckskin occurs naturally on sweet cherry in Contra Costa, Napa, Solano, and Sonoma Counties and on peach in Butte, Contra Costa, Glenn, Merced, Napa, Placer, Santa Clara, Solano, Sutter, and Tehama Counties, Calif. Various strains of the virus are rather widely distributed in the United States, where they cause disease on peach, cherry, or chokecherry.

Economic Importance

Because of the wide geographic distribution of the various strains of this virus and the severe loss that they cause over wide areas, buckskin is probably the most important virus disease of sweet cherry and among the most important virus diseases of peach. Numerous sweet cherry orchards in California have been removed because of the disease.

Host Range and Varietal Susceptibility

Sweet cherry (*Prunus avium*) varieties Chapman, Black Tartarian, Bing, Lambert, Napoleon (Royal Ann), Black Republican, and Rockport are susceptible to natural infection. Varieties that appear to show some resistance to natural infection are Long Stem Bing and Coop's Special. Peach (*P. persica*) varieties known to be susceptible to natural infection in California are Early Crawford, Elberta, Fay Elberta, Lovell, Muir, Orange Cling, Phillips, Shalil, Stuart, and Tuskena (Tuscan). Species that have been artificially infected are western chokecherry (*P. virginiana* var. *demissa*); sour cherry (*P. cerasus*) varieties English Morello, Stockton Morello, and Early Richmond; bitter cherry (*P. emarginata*); mahaleb cherry (*P. mahaleb*); almond (*P. amygdalus*); and smoothpit peach (*P. mira*). Various hybrids (*P. persica* × *P. amygdalus*, *P. mira* × *P. persica*, *P. davidiana* × *P. amygdalus*, and *P. amygdalus* × *P. fenzliana*) also have been artificially infected.

In preliminary tests desert peach (*P. andersonii*), myrobalan plum (*P. cerasifera*), David peach (*P. davidiana*), domestica plum (*P. domestica*) variety French (Agen), fenzl almond (*P. fenzliana*), desert apricot (*P. fremontii*), hollyleaf cherry (*P. ilicifolia*), and klamath plum (*P. subcordata*) were nonsusceptible.

Symptoms

Sweet cherry.—The symptoms of buckskin on sweet cherry on mazzard rootstock (*P. avium*) are very different from those on the same varieties on mahaleb (*P. mahaleb*) rootstock (*180*). There are at least three strains (Green Valley, Napa, and Palo Alto) and probably more of the buckskin virus in California alone. These strains can be distinguished by the characteristic symptoms they produce on sweet cherry on mazzard rootstock or on peach, and therefore these hosts are most useful in the identification of the disease. The symptoms on trees on mahaleb rootstock are so similar to those produced as a result of girdling by gophers and various undetermined causes as to make them of less diagnostic value. Infected trees on mazzard rootstock ordinarily live for many years after infection, whereas those on mahaleb rootstock quickly show a marked decrease in vigor and often die within 1 or 2 years after infection.

The symptoms produced by the Green Valley strain on sweet cherry on mazzard rootstock are usually localized at first, but after several years they occur throughout the trees. Fruits fail to mature, remain small, have short pedicels, hang on the tree after normal cherries have dropped, and tend toward a conical form (pl. 14, *A, B*). Healthy fruits are shown in plate 14, *C* and *D*. The blossom-end half of a fruit loses its natural luster and has the characteristic buckskin appearance. Dark-colored fruits remain red when they would normally be of a darker color; light-colored fruits remain yellowish when they would normally have some red color at maturity. Affected trees appear nearly normal during most of the growing season, but they may show a faint bronzing of the leaves, retarded growth, and dieback after several years of infection. During late summer or early fall the leaves have an orange-red color along the basal portion of the midrib and in the adjacent lamina (pl. 14, *G*). Infected trees can accordingly be most readily detected at two periods in the season, namely, when normal fruits mature and in the early fall, when leaves of infected trees have the orange-red color along the base of the midribs.

The symptoms produced by the Napa strain on trees on mazzard rootstock differ from those produced by the Green Valley strain in the following respects. On the affected portions of the trees the fruits fail to mature but drop about ripening time. The fruits do not lose their luster or have the conical form characteristic of infection with the Green Valley strain. The pedicels are of normal length. The tree symptoms differ in that most spurs produce terminal growth with short internodes. Such growths carry an excess number of small pale-green leaves and therefore have the rosetted appearance shown in figure 37, *C*. The buds on affected portions have loose scales and a resultant ragged appearance.

When sweet cherry trees are on mahaleb rootstock the symptoms produced by the two strains of virus are very similar. The first symptom observed is a general chlorosis of the tree followed by an upward rolling of the leaf edges. The leaves begin dropping in midsummer, usually showing an orange-red color along the midrib and lateral veins just before abscission. Much of the mahaleb root system of an infected tree usually dies rather quickly after infection. If a tree survives into the second year, it usually has sparse foliage and small pale-green leaves (fig. 37, *A*). A healthy branch is shown in figure 37, *B*. Infected trees produce few fruits. These ripen and are approximately normal in appearance, but are soft and insipid. Infection with

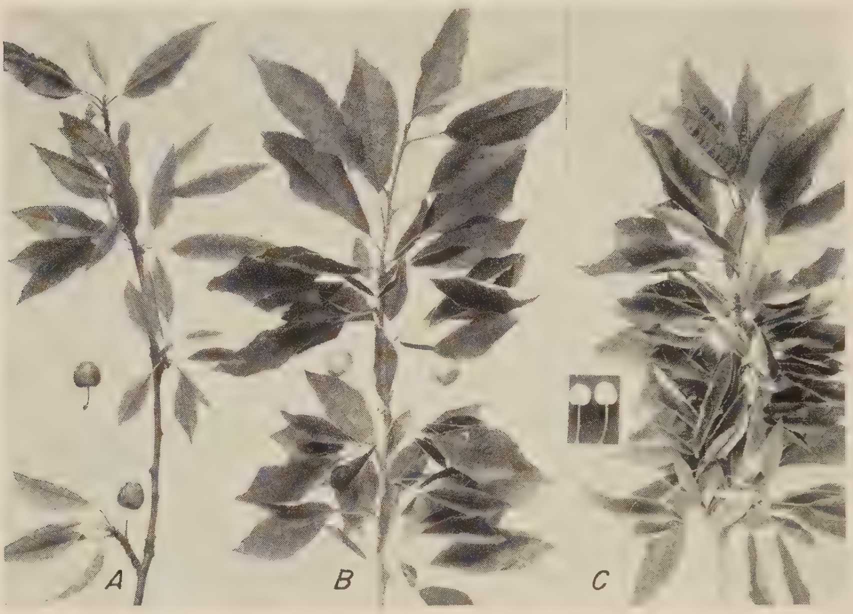

Figure 37.—*A*, Branch of Napoleon cherry on mahaleb rootstock, showing the sparse foliage characteristic of infection with the Napa strain of buckskin virus; *B*, unaffected Napoleon cherry branch; *C*, branch of Napoleon cherry on mazzard rootstock, showing the rosetted foliage characteristic of infection with the Napa strain of buckskin virus.

the Napa strain occasionally causes the fruits on certain small branches to remain small, immature, and green. These branches usually die before the end of summer. June buds taken from these branches produce infection when used as inoculum.

Schneider (*202*) found that there are much injury and wound-gum accumulation in sieve tubes of infected trees on mahaleb rootstock. These are most evident in the tissues near the union. The wound gum becomes red when sections are treated with phloroglucinol and hydrochloric acid. The same reaction occurs in the branches of infected peach trees.

Peach.—Peach trees infected with the Green Valley strain show the following symptoms on certain branches. Leaves are pale green to greenish yellow; those on older portions of affected branches have upward-rolled edges and are recurved toward the stem. On the more vigorous apical portions of affected shoots the leaves ordinarily appear normal until May or June, when few to many red or purplish blotches appear in the leaf blade. These blotches soon become necrotic (pl. 14, *F*) and drop out, producing a ragged appearance of the leaves. Leaves drop from such branches in early summer to midsummer, in succession from the base of the shoot upward. The fruits on affected branches shrivel and drop early. Ordinarily the symptoms are seen first on one or more branches and gradually from year to year symptoms appear on additional branches until most or all of them are affected. Affected limbs often die back; when such branches are removed a tree may appear healthy for a year or more before it again shows symptoms.

On peach the Napa strain produces symptoms similar to those produced by the Green Valley strain. The main difference appears to be that the symptoms produced by the Napa strain are much milder and tend to disappear the second year after infection.

A third strain in California, known as the Palo Alto strain (*202*), causes continued slow growth of affected peach branches throughout the summer. Such branches have very short internodes and leaves that are much smaller than normal.

Sour cherry.—English Morello trees on mazzard rootstock when inoculated with the Green Valley strain of the buckskin virus produced little terminal growth. The fruits remained small, and they shriveled before ripening. Early Richmond trees on mahaleb rootstock showed a reddening of the leaves in June, but the green color persisted in a narrow band around the margin of the leaf. Such leaves dropped early.

Western chokecherry.—Western chokecherry infected with buckskin virus shows a conspicuous carmine color on the lower leaves (pl. 14, *E*). This color is more or less continuous along the edges of the leaves and between the main lateral veins. The upper side of the midrib, the main lateral veins, and the adjacent tissues tend to remain green.

Transmission and Natural Spread

Transmission has resulted from use of diseased scions or buds from certain infected hosts. Best results are obtained by use of scions from infected Napoleon cherry trees growing on mazzard rootstock. Scions of Napoleon and other varieties of sweet cherry tried on mahaleb rootstock do not provide effective inoculum. Peach scions are also rather poor inoculum.

It is suspected that the virus is naturally transmitted by insects, but numerous attempts to transmit it by such means have been unsuccessful.

Under natural conditions the Green Valley strain of the virus spreads readily from cherry to adjacent peach orchards. Such spread has not been observed in the case of the Napa strain of the virus in a number of localities where peaches are adjacent to heavily infected cherries. One instance of infection of peach in Napa Valley has been seen in a comparatively isolated location, but the symptoms resembled more closely those of the Green Valley disease. The peach may be readily infected with the Napa strain by grafting. It is therefore possible that the absence of natural spread of the Napa strain from cherry to peach may be due to the absence of a suitable insect vector in Napa Valley.

Incubation Period

Cherry trees inoculated by grafting in February may show symptoms by the following June. On peach trees grown in the greenhouse the incubation period may vary from two to many months.

Control Measures

Sweet cherry trees produced by top-working the desired variety on several scaffold limbs of mahaleb seedling rootstock have been commercially profitable in the locality where the Green Valley strain occurs. Where infection occurs on individual arms the virus apparently fails to pass to other arms through the mahaleb (*179*) and the diseased arms can be removed. Top-working has therefore been the most satisfactory form of control for the Green Valley strain of the virus on sweet cherry. Most sweet cherry varieties do not make an entirely satisfactory union with mahaleb, and this incompatibility has caused some difficulty. Bing makes a good union with mahaleb, and double-working may therefore prove to be a solution for this difficulty.

A satisfactory control for the Napa strain on sweet cherry has not been worked out. In several States removal of infected chokecherry has been recommended as a control measure for X-disease (*103, 226*).

ALBINO

By S. M. Zeller, C. B. Cordy, and J. A. Milbrath

Name of the Causal Virus

Albino virus.

Geographic Distribution

The disease was first discovered in Ashland, Oreg., in June 1937 (*266*). The local spread of albino has been very rapid, but the known distribution is limited to the Rogue River Valley in southern Oregon.

Economic Importance

Albino is of great economic importance because it spreads rapidly and kills affected trees. It has already eliminated several small orchards in the district where it was first observed and will eventually eliminate commercial cherry production in the Rogue River Valley. In one orchard without previous infections, 38 percent of the trees were diseased in the first year of infection and the following year over 90 percent were infected (*67*). Should this disease become generally distributed the losses would be great.

Host Range and Varietal Susceptibility

The disease affects sweet cherry (*Prunus avium*) on mahaleb (*P. mahaleb*) or mazzard (*P. avium*) rootstocks. Albino has been found on Bing, Black Republican, Lambert, Black Tartarian, and Napoleon (Royal Ann) varieties in orchards. The Bing, Napoleon, and Black Republican varieties are all severely affected. Napoleon is possibly the most severely affected and most rapidly killed of these three varieties. The Lambert variety appears able to endure the disease longer than other sweet cherry varieties so far found affected with albino, but it gradually succumbs to the disease. Black Tartarian shows considerable resistance. Montmorency sour cherry (*P. cerasus*) has been experimentally infected.

Symptoms

All varieties of sweet cherry affected with albino show approximately the same symptoms. Lambert, however, may be severely affected without showing the typical fruit symptoms. In orchards the disease kills Bing and Napoleon varieties more quickly than others. All or any portion of the tree may be affected with dieback, a usual symptom on diseased branches, beginning in early spring as soon as leaves appear. As early as June leaves become uniformly golden bronze green to olive brown, and the margins roll upward (pl. *15*, *B*, *C*). Occasionally leaves about to be shed become bright yellow to orange, with some pinkish tints, especially in a pinnate pattern along the midrib and lateral veins at the base. In late summer new growth of small, green, rosetted leaves is produced from terminal buds (pl. 15, *C*). This character is general on moderately diseased trees, and occasionally it is the first symptom observed on otherwise vigorous trees. Fruits remain small and immature, even those of dark-red-fruited varieties turning white (pl. 15, *D–F*). A heavy drop may occur before harvesttime, but some of these small white fruits remain attached long after normal fruits have dropped.

In the experimental nursery certain varieties infected by inoculation reacted differently from the way they did in the orchard. (See Transmission.)

Transmission

Nursery trees of Bing, Napoleon, Lambert, Black Republican, and Centennial sweet cherries and Montmorency sour cherry on mahaleb and mazzard rootstocks in separate series were infected with albino virus by inoculation in the fall with buds from diseased shoots or by grafting with spurs which had borne albino fruits. In the same experiment infection was obtained on Black Tartarian trees growing on mahaleb rootstock.

When the trees were on mahaleb rootstock, all varieties except Black Tartarian and Montmorency were killed by albino during the summer after inoculation (pl. 16, *A*). In contrast, when the trees were on mazzard rootstock, most of them showed a severe shock reaction the first year, but only a few died. Many of them were still alive 3 years after inoculation. At that time the trees were dwarfed, the nodes were so shortened that the growth was rosetted, and the foliage was light in color, often with a bronze cast. All these trees produced albino fruits. Black Tartarian trees on mahaleb rootstock reacted just as other varieties did on mazzard rootstock, thus indicating that this variety has some resistance to albino.

Montmorency sour cherry on both rootstocks showed considerable resistance to albino. All inoculated trees developed some symptoms of shock, but none died. Those on mazzard rootstock had a somewhat more severe reaction than those on mahaleb rootstock, possibly indicating that sour cherry on mahaleb does not react in the same way as sweet varieties do. Three years after inoculation the trees had declined and died back somewhat. Occasionally fruits on the most severely affected trees were small and light-colored, resembling the albino fruits of sweet cherry varieties.

No fruit or leaf symptoms were produced on peach varieties inoculated with buds from sweet cherry trees affected with albino in numerous tests. Indexing from the inoculated peach to sweet cherry indicated that peach is immune from the albino virus. In a recent experiment alternate trees in a block of 90 young peach trees of 7 varieties were inoculated in the fall with buds from sweet cherry trees affected with albino. The summer after inoculation all uninoculated trees were normal green, but all inoculated ones had developed a chlorosis similar to that induced by lime. Whether the reaction was caused by albino virus or by ring spot virus as a contaminant in the inoculation is not known.

Incubation Period

Sweet cherry trees bud-inoculated in late summer showed symptoms within 10 months.

Control Measures

No particular control measures have been outlined. Since albino is of limited occurrence in a section where cherries are not of much commercial importance, eradication should doubtless be attempted. Until such eradication is realized, measures should be taken to prevent the distribution of cherry scion wood from the section where albino exists.

Remarks

Several of the symptoms described for albino are similar to those of buckskin (*180, 257*). Among these are the small whitish fruits, the pinkish and orange tints at the base of leaves extending up the midvein and pinnately into the lateral veins, and the soft buds and stimulated growth of small rosetted green leaves late in the growing season. Certain characteristics, however, distinguish albino from buckskin. The uniform golden-bronze greenness of the leaves, which is generally found associated with albino, is not described as a symptom of buckskin.

In orchards sweet cherry varieties are killed by the albino virus (pl. 15, *A*), but experimentally infected nursery trees reacted differently on mazzard rootstock from the way they did on mahaleb. Cherries on either rootstock are naturally affected with Green Valley buckskin, according to Rawlins and Parker (*179*), but they develop different symptoms; they wilt and die when on mahaleb rootstock, whereas they produce small buckskin cherries but have no leaf chlorosis during the growing season when on mazzard. Western X-disease (leaf-casting yellows) (*234*) of peach has been recovered from cherry trees affected with Green Valley buckskin in California (*180*) and from diseased cherry trees bearing little fruits in eastern Oregon; but, although there are many peach trees interplanted with the cherries in the section where albino occurs, no virus disease of peach has been observed there. Peach trees inoculated with the albino virus have not developed any known virus disease. Therefore, Green Valley buckskin, which seems to be caused by the virus that causes western X-disease of peach, is distinct from albino.

MOTTLE LEAF

By H. R. McLarty, T. B. Lott, J. A. Milbrath, E. L. Reeves, and S. M. Zeller

Common Names of the Disease

The common name "cherry mottle leaf," which has come into general use in the Pacific Northwest, was first applied by Zeller (*255*) in 1934. Zeller and Milbrath gave the name "severe mottle leaf" to the rugose type and "mild mottle leaf" to the smooth type.

Names of the Causal Virus

The following names have been applied to the virus: "*Marmor cerasae* Zeller and Evans" (*259*), "*M. cerasi* (Zeller and Evans) Holmes," "Prunus virus 7 Zeller and Evans" (*259*), and "mottle leaf virus." Under the system of naming proposed by Fawcett the name would be "*Prunivir cerasi.*"

History and Geographic Distribution

Severe mottle leaf was first noticed in Oregon by Zeller (*255*) in 1920. The virus nature of the disease was established in 1935 by McLarty (*146*) and Reeves (*181*). The disease occurs generally in the sweet-cherry-growing districts of Washington, Oregon, and Idaho and of British Columbia, Canada, and has been observed in California, Utah, and Montana. In Washington severe mottle leaf is most prevalent in the foothill or canyon orchards where the native wild bitter cherry (*Prunus emarginata*) may be found growing nearby, and it occurs less frequently in open, wide valleys. In these latter locations mild mottle leaf is the prevailing type.

Surveys conducted by Coe (*66*) showed that 1.4 percent of the total bearing sweet cherry trees in Washington and 3.5 percent of the symptom-expressing varieties were affected with mottle leaf. In Oregon mild mottle leaf is prevalent in all districts where sweet cherries are grown, and severe mottle leaf is found occasionally, as if introduced in grafting wood or nursery stock.

Economic Importance

Severe mottle leaf is the most important cherry disease in certain foothill or canyon districts of north-central Washington. It is comparatively minor elsewhere in Washington and in California. In Oregon certain forms of mild mottle leaf constitute an important factor in cherry production. In British Columbia the disease is considered of importance in the Kootenay area, where the severe mottle leaf type predominates, and of minor importance in the Okanagan, where mild mottle leaf is the prevailing type. In favorable locations mottle leaf is considered to be a serious disease of sweet cherries.

Host Range and Varietal Susceptibility

Bing and Napoleon (Royal Ann) are the two varieties of sweet cherry (*P. avium*) most severely affected, but other varieties are susceptible and suffer injury in varying degrees. Sour cherry (*P. cerasus*) and duke cherry (*P. avium* × *P. cerasus*) are not immune, but they are practically symptomless carriers. Reeves (*183*) studied the reactions of various species and varieties and reported the relative intensity of mottle leaf symptoms, shown in table 2.

TABLE 2.—*Relative intensity of mottle leaf symptoms on species and varieties of cherry*

Prunus species	Varietal or common name	Leaf symptoms	
		Mottling	Rugosity
P. avium	Bing	Pronounced	Severely puckered.
	Napoleon	do	Do.
	Waterhouse	Moderate	Puckered.
	Centennial	do	Do.
	Black Tartarian	Slight, variable	Moderately ruffled.
	Early Purple	do	Do.
	Governor Wood	do	Do.
	Black Republican (Republican).	do	Do.
	Lambert	Slight, fugitive	Lightly ruffled.
	Deacon	do	Do.
	Parkhill seedling	do	Do.
P. cerasus	Montmorency	Occasional, slight	None.
	Early Richmond	do	Do.
	English Morello	None	Do.
P. avium × *P. cerasus*	May Duke	Occasional, slight	Lightly ruffled.
	Late Duke	Very slight, fugitive	Occasional light ruffling.
	McKee	do	Do.
P. mahaleb	Mahaleb cherry	Occasional, slight	Do.
P. emarginata	Wild bitter cherry	Medium, fugitive	Do.

Zeller and Milbrath found that the severity of reaction of sweet cherries to both severe mottle leaf virus and strains of mild mottle leaf virus was in the order: Bing, Napoleon, Black Republican, and Lambert. Some mazzard seedlings showed violent reaction to severe mottle leaf virus. Peaches (*P. persica*) apparently were symptomless carriers of both severe and mild mottle leaf virus, but this could not be definitely determined because of the interference of latent sweet cherry viruses. Black Republican and Lambert varieties, when inoculated with mild strains of the mottle leaf virus, were essentially symptomless except for an occasional chlorotic, elongated spot parallel with the lateral veins.

Symptoms

The most distinctive symptoms of mottle leaf are expressed on the leaves of affected trees. When severe mottle leaf occurs on the most susceptible varieties, Bing and Napoleon, there are an irregular, chlorotic mottling and a distortion early in the season and increased puckering of the leaves and variable mottling as the season progresses (fig. 38, *A, B*). Leaves are often reduced in size and show some variable lacerations or shot holes following necrosis, but there is no leaf casting (fig. 38, *C*). When leaf symptoms are severe the fruits may be abnormally small, later than normal in ripening, and insipid in flavor, but not misshapen. The growth of the tree is stunted; the stunting brings the spurs close together and gives the tree a rosetted appearance. On other varieties the symptoms are generally similar in nature to those on Bing and Napoleon, but they are less pronounced and vary widely from variety to

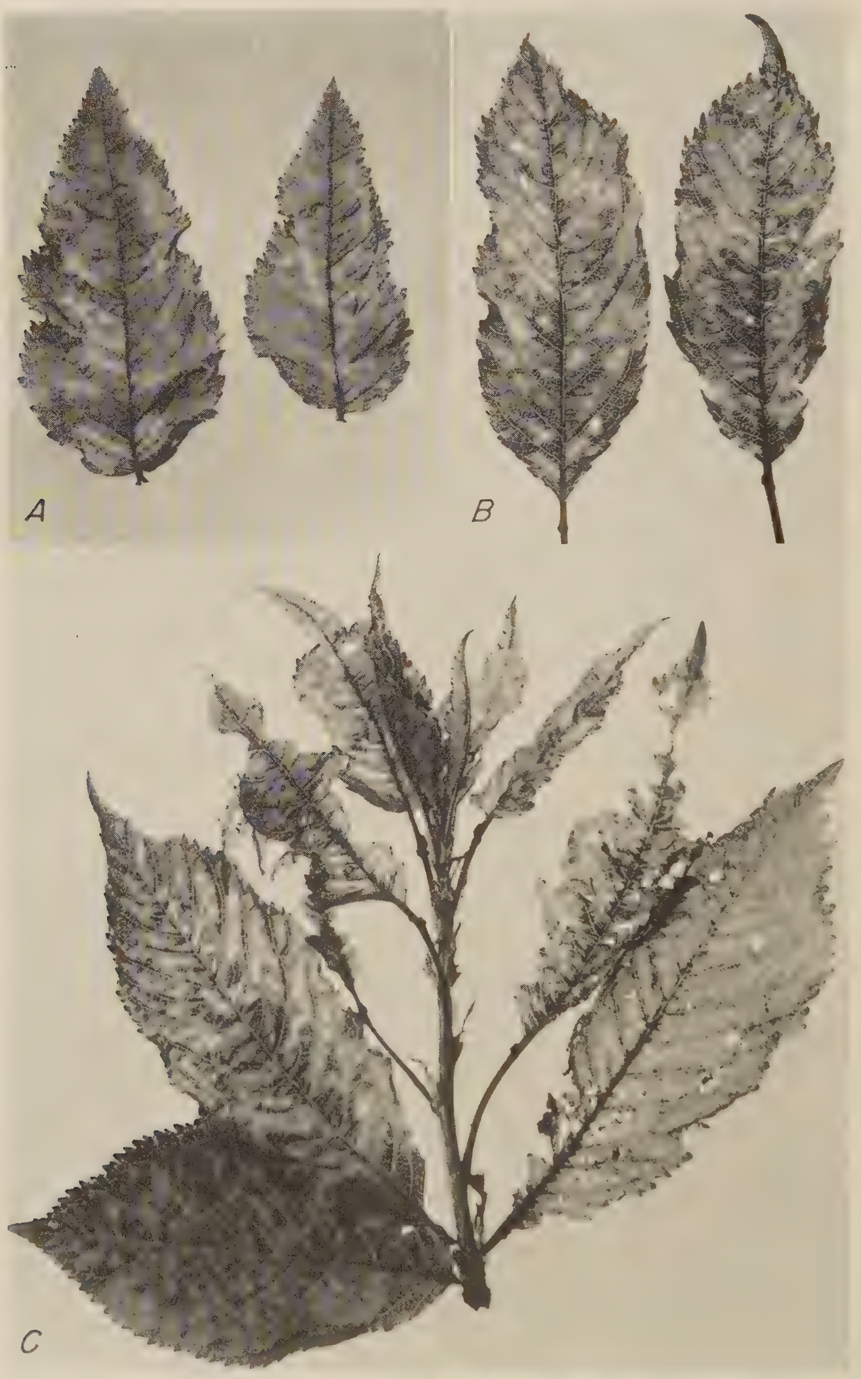

Figure 38.—Cherry leaves affected with severe mottle leaf: *A*, Napoleon; *B*, Bing. *C*, Branch of Bing cherry affected with severe mottle leaf, showing symptoms in the new tip growth.

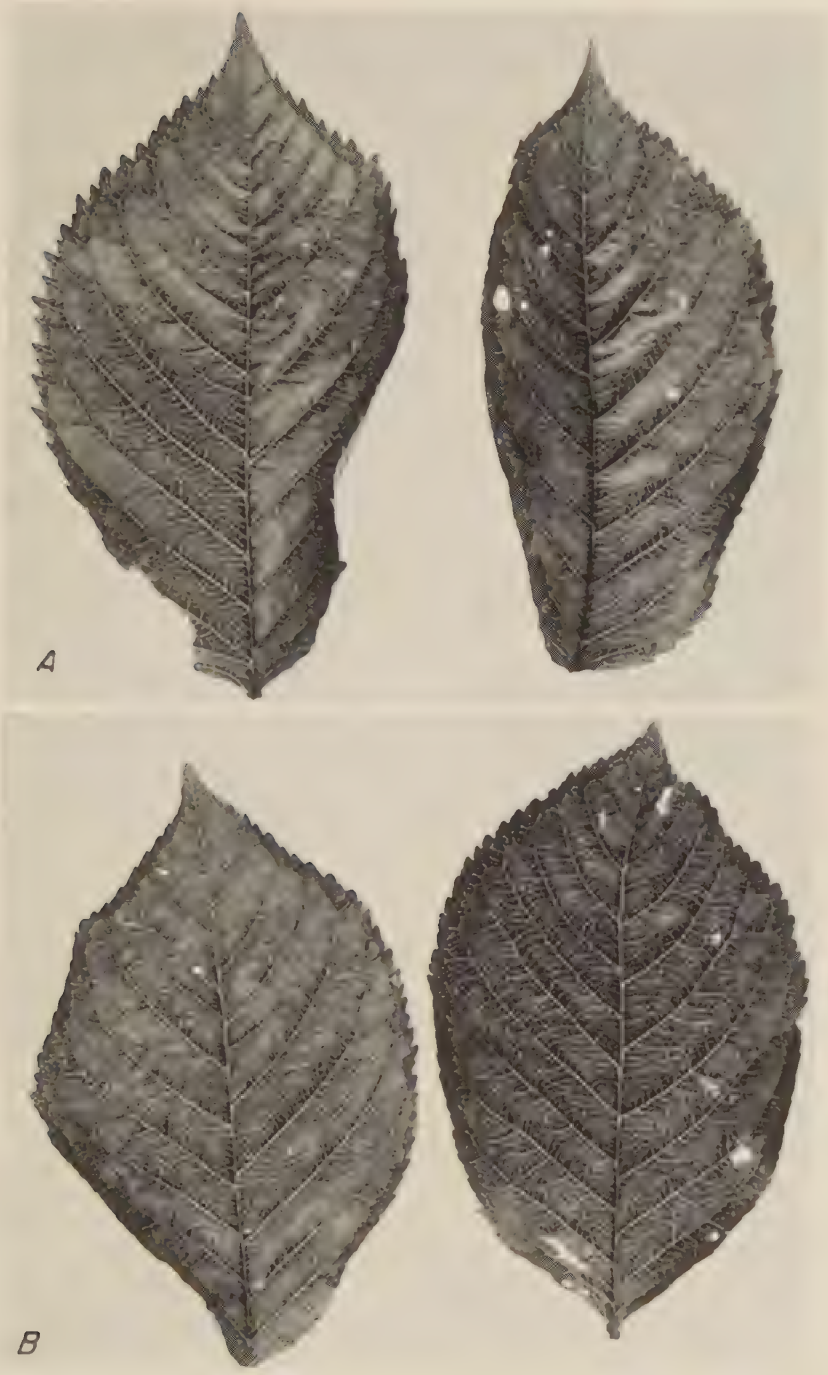

Figure 39.—Cherry leaves affected with severe mottle leaf: *A*, Black Republican; *B*, Lambert.

variety (fig. 39, *A, B*). Mild mottle leaf virus causes similar symptoms of less intensity (fig. 40), and tree productivity is not materially affected (*184*). The different strains appear to be stable, inducing their characteristic effects through subsequent transfers without attenuation or increment of symptoms.

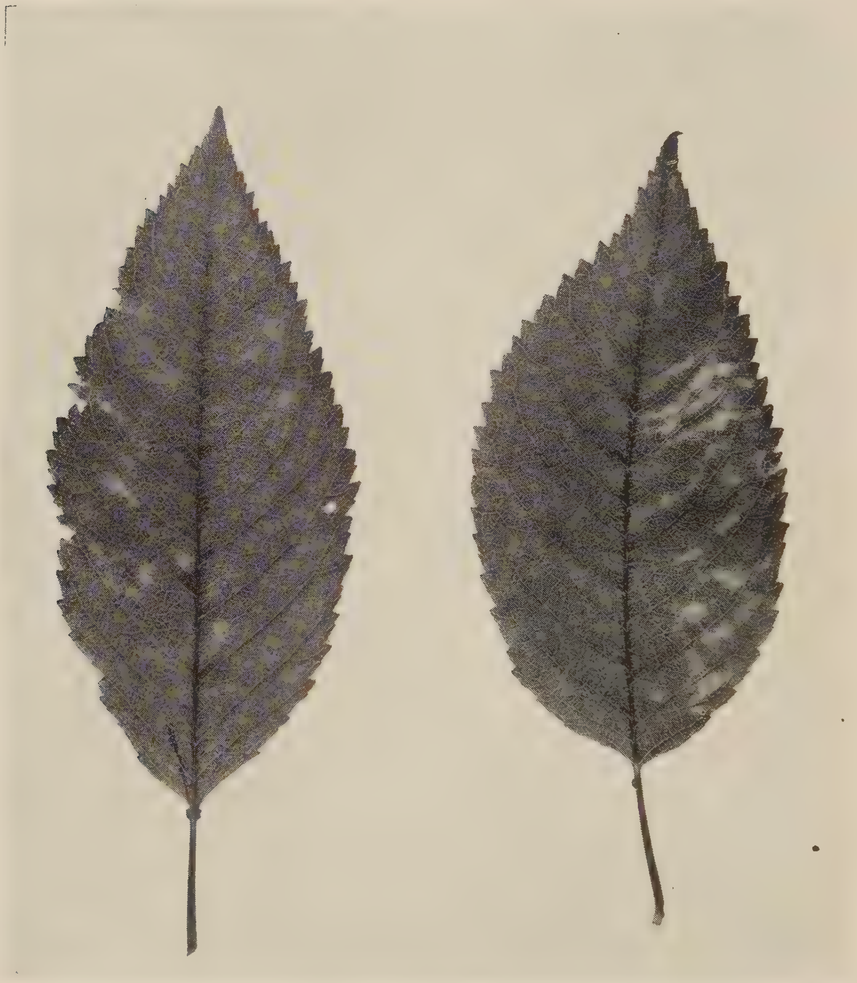

***Figure 40.*—Napoleon cherry leaves affected with mild mottle leaf.**

Zeller and Milbrath found that inoculations with mild mottle leaf virus caused considerable dwarfing of young Bing and Napoleon trees. In Oregon, orchard trees infected by these mild strains showed loss of vitality and much dieback. It was felt that some of the dieback might have been due to an induced sensitivity brought about by drought, winter injury, or other factors.

Symptoms of both severe and mild mottle leaf are at least partially masked by high temperatures. Masking is indicated by the occurrence of characteristic symptoms on new growth in the spring or fall and the absence or suppression of these symptoms on new growth in midsummer.

Transmission and Incubation Period

Transmission is readily obtained through any tissue union that results from budding or grafting. The natural vector of the virus is not known.

There is some evidence to indicate that the virus spreads from the wild bitter cherry to nearby cherry orchards. Surveys showed that there was a higher incidence of mottle leaf in orchards located close to native stands of this cherry. Spread in an orchard varies widely in different localities and in different years. Reeves (*183*) stated that in north-central Washington in 52 orchards in which at least 1 affected tree was known to be present the spread over a 4-year period was considered serious in only 7 orchards. In 1 planting of 84 trees 6 showed mottle leaf in 1935; 1 new infection occurred in 1936, 0 in 1937, and 21 in 1938.

Zeller and Milbrath transmitted both severe and mild mottle leaf virus from cherry to peach and back to cherry. In limited tests they showed that the virus is not seed-borne.

In artificial transfers the period of incubation varies according to the stage of seasonal development. Reeves (*183*) reported that in spring inoculations in the field symptoms were obtained as early as 37 days after treatment, whereas those made in midsummer and later failed to produce symptoms until the following spring. In greenhouse tests symptoms were obtained after 14 days.

Control Measures

Control of mottle leaf in future orchards must be through the use of clean nursery stock or grafting wood. For all propagation work scions should be obtained only from trees that have been proved to be virus-free.

Since trees affected with this disease do not recover, diseased-tree removal is recommended in established orchards when productiveness is affected. In orchards adjacent to stands of wild bitter cherry it is recommended that tolerant sweet cherries be used. It is not considered practical to eradicate the wild bitter cherry.

Remarks

The name "mottle leaf" is used to designate a diseased condition caused by several distinct but closely related virus strains. The symptoms they induce in infected plants are classified generally into two groups, severe mottle leaf and mild mottle leaf. All strains of the causal virus may be transmitted without change in type of symptom expression.

RUSTY MOTTLE [22]

By E. L. Reeves

Other Common Names of the Disease

Zeller and Milbrath (*264*) referred to rusty mottle as severe rusty mottle.

Names of the Causal Virus

Marmor rubiginosum Reeves (*182*) has been used. Under the system of naming proposed by Fawcett the name would be "*Prunivir rubiginosum.*"

History and Geographic Distribution

In 1940 rusty mottle was reported as a new virosis of cherry that had been repeatedly transmitted during the previous 5 years by grafting or by some adaptation of the grafting process (*182*). It was also referred to as a disease sometimes confused with mottle leaf of cherries (*183*). In 1942 Zeller (*256*) referred to rusty mottle in Oregon as a bronzed ring spot type of disease differing from the type described in Washington. In 1947 Zeller and Milbrath (*264*) further described the type occurring in Oregon as mild rusty mottle, made comparative studies of it and the type described by Reeves (*182*), and decided that the two types were distinct in several respects. In 1945 Richards and Rhoads (*200*) described a rusty mottle of the sweet cherry in Utah, now referred to as necrotic rusty mottle (p. 120), which has some symptoms closely resembling those of the disease that occurs in Washington but which affects the tree more seriously. Later Reeves and Richards (*189*) described certain differences between the types of rusty mottle in Utah and Washington, but in the absence of comparative tests reached no positive decision as to their relationship.

Reeves (*184*) stated in 1943 that "different strains of the virus may be involved in the problem . . .," but during an orchard survey in Washington, reported by Coe (*66*), no distinction was made in reporting rusty mottle as it was first described and the supposedly milder form of the disease. It is now recognized that probably more than half of the rusty mottle reported in the 1942 orchard survey (*66*) is the same as mild rusty mottle described by Zeller and Milbrath (*264*). Also, during 1946 in one Washington orchard a diseased cherry tree was observed with symptoms more closely resembling those of necrotic rusty mottle in Utah than any disease previously observed in Washington. Similar symptoms were observed on several trees in two other orchards near Wenatchee, Wash., in 1949.

Rusty mottle has been found distributed in cherry orchards of Chelan, Douglas, and Yakima Counties, Wash. In Idaho diseased cherry trees with symptoms closely resembling those of rusty mottle have been observed, but no positive statement can be made whether the disease is identical with rusty mottle.

Economic Importance

Although rusty mottle appears to be somewhat localized in its occurrence in Washington cherry orchards, it is of economic importance in the orchards where it is found. Orchard surveys in Washington, reported by Coe (*66*), showed that 59 out of 810 properties inspected in 3 counties had trees affected with rusty mottle. While the average of infected trees in the 59 orchards was only 2.9 percent, a few orchards had 19.4 to 34 percent of the trees affected. However, as previously stated, in the survey reported by Coe (*66*) no distinction was made between rusty mottle and mild rusty mottle. How many of the trees were actually affected with rusty mottle as described in this section can only be estimated; possibly about 25 to 30 percent were so affected.

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[22] The report given in this section is confined to the type of rusty mottle originally described by Reeves (*182*).

Host Range

In Washington all varieties of sweet cherry (*Prunus avium*) commonly grown in commercial orchards and mazzard (*P. avium*) seedlings have been found affected or have been successfully inoculated by budding. Several varieties of peach (*P. persica*) and sour cherry (*P. cerasus*) have been successfully inoculated by budding, but they have not been found naturally infected.

Symptoms

Sweet cherry.—In Washington Reeves (*182, 183*) reported that the first foliage symptom appears 4 to 5 weeks after full bloom as a chlorotic mottling of the older leaves. All the foliage soon becomes more or less affected, and various percentages of the leaves develop late-season colors (bright yellow to red) with islands of green (fig. 41, *B*). Leaf casting of the brightly colored foliage takes place largely during the 2 to 3 weeks prior to fruit harvest. The mottling of the remaining foliage then becomes more pronounced and the chlorotic spots and areas become yellowish brown; and a general rusty appearance of the foliage results. On trees affected for more than 2 years fruits are often smaller than normal ones, retarded in maturity, and insipid in flavor, but not misshapen (fig. 41, *C*). The general effect of the disease on the fruits is apparently dependent upon the severity of foliage symptoms and the percentage of leaves cast prior to harvest.

Peach.—Reeves observed foliage symptoms on several varieties of freestone peach inoculated with buds from cherry trees affected with the type of rusty mottle reported in Washington. Symptomatological patterns on peach leaves are variable and striking in appearance. During late June and early July a variable portion of the foliage turns partially greenish yellow, yellow, and sometimes orange, and the chlorophyll degenerates so as to produce various ring spots and patterns. Such affected leaves soon fall, and relatively few leaves with distinctive recognizable symptoms remain on the tree. The period during which recognizable foliage symptoms may be observed is variable, but it may be as short as 12 to 15 days.

Transmission

Transmission has been easily effected by means of grafts or some adaptation of the grafting process with tissues taken from either shoots or roots of a diseased tree.

Incubation Period

Trees inoculated in late summer by budding exhibit symptoms the following year at the time when symptoms of the disease usually appear. Trees inoculated during late March and early April by top or root grafting often exhibit symptoms by mid-June; but sometimes recognizable symptoms do not appear until the following year.

Control Measures

Diseased trees should be removed if they are affected so much that a profitable crop is no longer being produced or whenever their presence is considered a menace to the healthy trees in the orchard. The careful selection of propagation wood from nonaffected trees is especially recommended as a preventive measure.

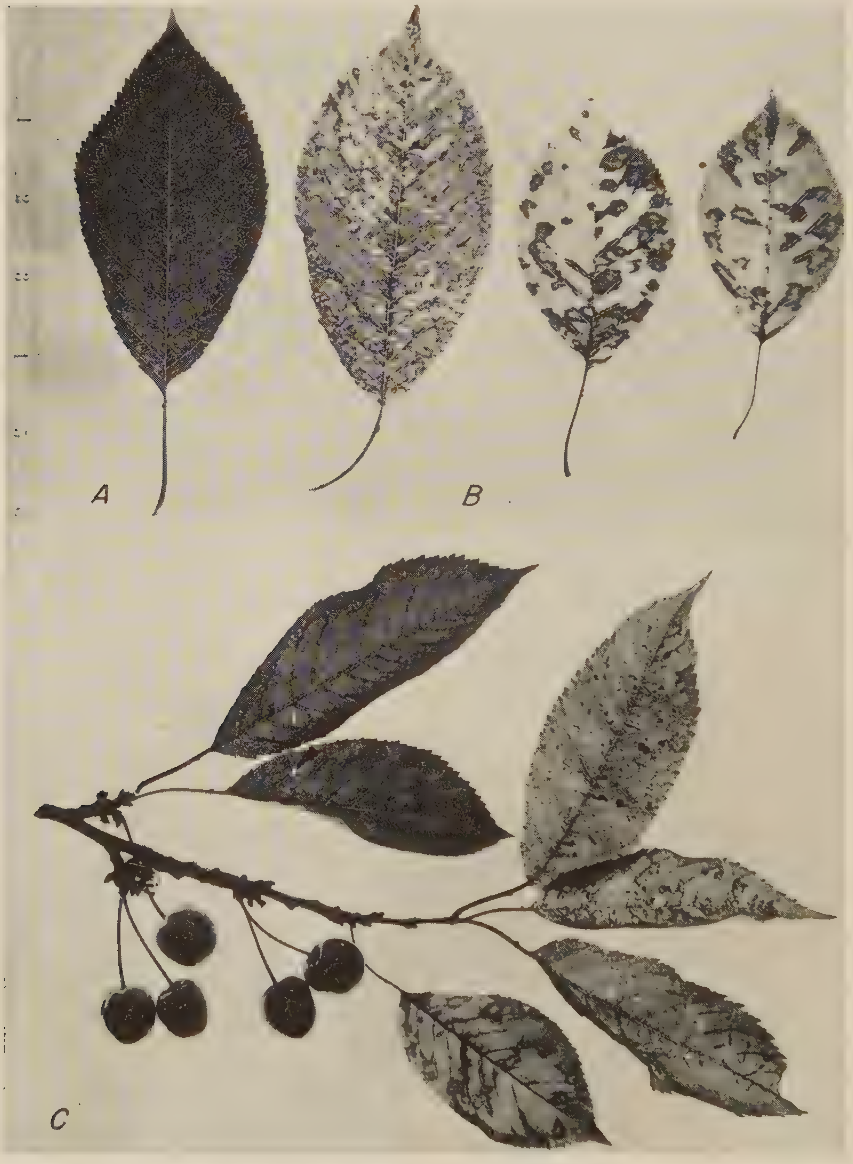


Figure 41.—*A*, Normal leaf of Bing cherry; *B*, comparable leaves affected with rusty mottle, showing various patterns that develop on leaves that are cast prior to harvesttime; *C*, branch from a Bing cherry tree affected with rusty mottle, 1 week before harvesttime, showing that the fruits are not malformed but that the leaf cast has been heavy.

Remarks

While the leaf patterns as exhibited by the affected foliage (fig. 41) are variable, there is some resemblance between these symptoms and those exhibited by foliage of sweet cherry affected with each of at least three other virus diseases: namely, mild rusty mottle (*264*), Lambert mottle (*141*), and a rusty-mottle-like virus disease of the sweet cherry in Utah (*189*), now referred to as necrotic rusty mottle. Under field conditions and at particular periods during the growing season leaves could be found on trees affected with any one of the four diseases that would exhibit symptoms common to the other three. However, there are other diagnostic features of each of the four. These are variable, and, of course, the intensity, extent, or severity of the leaf patterns of each disease may also vary. This emphasizes the necessity of further work in comparatively studying these four diseases and the impossibility of diagnosing accurately any of the four from a few detached specimens or from photographs of affected foliage.

MILD RUSTY MOTTLE

By S. M. Zeller and J. A. Milbrath

Name of the Causal Virus

Mild rusty mottle virus.

Geographic Distribution

Mild rusty mottle (*264*) occurs in some sections of Oregon, Washington, and Idaho. In Oregon it has been observed throughout the Willamette Valley and in Curry, Hood River, and Wasco Counties.

Economic Importance

Mild rusty mottle ranks first among the mottle-type virus diseases of sweet cherry in Oregon. Surveys (*256*) showed that 20 percent of the trees in one orchard at least 35 years old had mild rusty mottle and that in another orchard about 25 years old 32 percent of the trees were affected. In some orchards it was estimated that more than 50 percent of the trees were infected. On the other hand, some of the oldest orchards have little or no mild rusty mottle. Some young orchards are relatively free of it, but others, originating from different nursery stock, have scattered trees apparently infected from the start. Although trees affected with mild rusty mottle continue to produce fruit for a number of years, the trees gradually decline and the loss from the disease in northwestern sweet cherry orchards is undoubtedly of considerable economic importance.

Host Range and Varietal Susceptibility

Mild rusty mottle seems to affect one variety of sweet cherry (*Prunus avium*) about as much as another. In orchards it has been found affecting trees of Bing, Black Republican, Black Tartarian, Lambert, and Napoleon (Royal Ann) varieties. The causal virus has been obtained from Montmorency sour cherry (*P. cerasus*). A number of other species and varieties of stone fruits have been found by inoculations to be susceptible. Peach (*P. persica*) and Italian Prune (*P. domestica*) proved to be symptomless hosts of the virus. Mahaleb cherry (*P. mahaleb*) seedlings, western chokecherry (*P. virginiana* var. *demissa*), Indian plum (*Osmaronia cerasiformis*), and oriental flowering cherry (*P. serrulata*) varieties Amanogawa, Kwanzan, Shirotae (Mount Fuji), Naden, and Shiro-fugen were inoculated with mild rusty mottle virus, but slight if any symptoms were expressed. No return inoculations have been made to sweet cherry from the plants just listed.

Symptoms

Sweet cherry.—All varieties of sweet cherry affected with mild rusty mottle show approximately the same symptoms. Even from a distance affected trees stand out because of their general yellowish-green color. The foliage takes on a rusty or bronzed appearance by late June or early July. Older infected trees often show considerable dieback and unthriftiness, probably due to the combined effects of the disease and unfavorable factors such as drought and low temperatures. At any rate affected trees seem to decline more rapidly than healthy ones. Decline is indicated by poorer terminal growth, poorer set on fruiting spurs, and other symptoms, although it is much slower than that brought about by severe rusty mottle (rusty mottle, p. 112) (*182*).

During the first season of infection the disease may be found on only a single branch, but by the next season it may have spread to the whole tree. There are no particular leaf symptoms early in the spring. Usually the first mottling appears in May or June on leaves which have attained a certain maturity, some distance from the growing tips. There are no symptoms on the terminal growth throughout the season. The mottling starts as yellowish or light-green areas in the leaves. These areas are of various shapes (fig. 42).

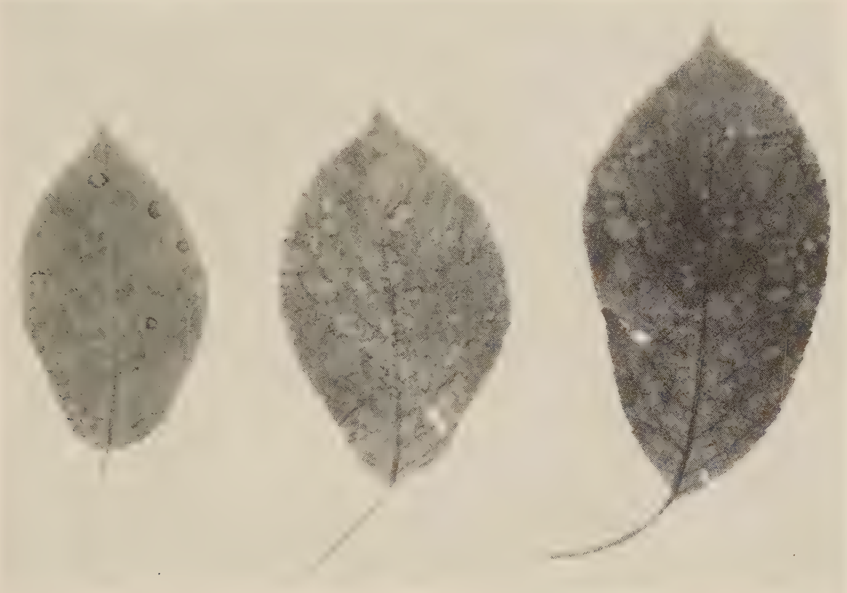


***Figure 42.*—Bing cherry leaves affected with mild rusty mottle.**

In individual areas the centers are lighter yellow than the margins, which gradually become bronzed or reddish. This bronzing or rustiness shows first on the upper leaf surface, but gradually the leaf becomes rusty below. Bronzing borders the areas whatever their shape or size. At times the bronzing or rustiness is in the form of tiny stipples. The stippling may be on the veins or very close to them before it shows in the interveinal tissue, and it usually starts at the base of the leaf blade. If the rustiness forms a line pattern or rings, it is usually feathered out on at least one side. At times the older leaves or those that are shaded, in the center of the tree, may become chlorotic and develop a bright-yellow or whitish mottle. Such leaves are shed early, but such shedding does not make the tree look generally defoliated. In unusual cases certain trees may shed many relatively greenish, mottled leaves even before harvesttime, but the next year the same trees may have no such leaf cast during the summer.

Fruits of infected trees are somewhat retarded in ripening. Fruits of the Napoleon variety are inclined to be clear yellow and usually without the red cheeks of healthy fruits.

Sour cherry.—Montmorency sour cherry trees from which mild rusty mottle was recovered had considerable dieback and a peculiar leaf symptom, neither of which has been definitely proved to be expressions of this particular disease. The leaves had a mild mottle, and they were much smaller than normal. The undersurface of the leaves was mottled with a rusty coloration brought about by necrosis of the lower epidermis in certain areas; the rustiness may be limited to one side of the leaf or to the central area or may occur along the veins. A close examination of the rustiness with a lens shows that it starts with a reddish necrosis along the veinlets. This spreads along these veins until the whole spot has a reddish-mottled appearance to the naked eye. At this stage the upper surface appears mottled, and the older chlorotic spots begin to show necrosis above.

Transmission

Mild rusty mottle is perhaps as easily transmitted as any other woody-plant virosis. Whenever sweet cherry was inoculated by budding with infected sweet cherry tissue, transmission was successful with or without apparent organic union of the two tissues. Inoculum from the following districts was compared on sweet cherry: Benton, Hood River, Lane, Multnomah, Polk, Wasco, and Washington Counties, Oreg., and Benton and Yakima Counties, Wash.

The disease was transmitted to the following varieties of sweet cherry: Bing, Black Republican, Deacon, Golden, Lambert, Napoleon, and Schmidt; to mazzard seedlings; and to Montmorency sour cherry. The following species or varieties showed slight if any symptoms: Mahaleb cherry seedlings; oriental flowering cherry varieties Amanogawa, Shirotae, and Shiro-fugen; western chokecherry; and Indian plum. Occasionally Kwanzan and Naden varieties showed slight leaf symptoms.

Many peach, plum, and prune varieties were inoculated, but none showed leaf symptoms. Peach and Italian Prune were proved by return inoculation to sweet cherry to be symptomless hosts of mild rusty mottle virus.

Incubation Period

Trees inoculated by budding or patch grafting in late summer showed symptoms in 10 to 12 months.

Control Measures

New orchards should be established with clean nursery stock. In established orchards where few trees are infected, the diseased trees should be removed. If the removal of infected trees would too greatly deplete an established orchard, it might be advisable to allow them to remain until unprofitable. If a thinning program is necessary in a too closely planted orchard, mapping of the diseased trees should precede pulling and the pulling of trees should be so arranged as to remove the greater number of diseased trees.

Remarks

Mild rusty mottle (*264*) is distinct from severe rusty mottle, which is caused by *Marmor rubiginosum* Reeves and was described by Reeves in 1940 (*182*) and 1943 (*184*). They differ in severity of effect on the trees, in symptom expression on leaves, fruits, and bark, and in the amount of leaf cast.

Sweet cherry trees affected with mild rusty mottle do not decline so rapidly as those affected with severe rusty mottle. The fruits of trees affected with mild rusty mottle are almost normal in size and quality, whereas those of trees of the same varieties affected with severe rusty mottle are reduced in size and insipid. The superficial splitting of the bark on 1-year-old wood caused by severe rusty mottle has never been observed on trees affected with mild rusty mottle.

Leaf casting of Bing cherry trees affected with severe rusty mottle is usually sufficient to cause the trees to have a stripped, bare, or defoliated appearance (*184*). Such severe leaf casting has never been observed in trees affected with mild rusty mottle.

Severe necrosis of the chlorotic areas in leaves of sweet cherry affected with severe rusty mottle is usually apparent by late spring or early summer; in trees affected with mild rusty mottle bronzing occurs, but necrosis is seldom if ever seen as a leaf symptom.

After about the first of June symptoms of severe rusty mottle show on leaves almost to the tips of branches, while on trees affected with mild rusty mottle there are no leaf symptoms for some distance back of the growing tips.

Certain plants, such as peach, western chokecherry, and some varieties of oriental flowering cherry, inoculated with mild rusty mottle virus, develop no particular leaf symptoms, whereas the same varieties and species inoculated with severe rusty mottle virus show the rustiness and bronzing in the leaves similar to those described on leaves of sweet cherry.

NECROTIC RUSTY MOTTLE

By B. L. Richards and E. L. Reeves

Other Common Names of the Disease

Rusty mottle (*195, 200*); rusty-mottle-like disease (*189*).

Name of the Causal Virus

Necrotic rusty mottle virus.

History and Geographic Distribution

The virus nature of necrotic rusty mottle of sweet cherry was not established until 1944 (*189, 195, 200*); however, the disease had been recognized many years earlier as a destructive factor in Utah orchards and had been referred to locally by such names as "cherry leaf spot," "cherry leaf drop," and "cherry leaf rust." In 1945 the disease was discussed in publications entitled "Rusty Mottle of the Sweet Cherry in Utah" (*195, 200*), because of certain similarities of the disease in Utah to rusty mottle of sweet cherry in Washington described by Reeves (*182*) in 1940. In 1946 the disease in Utah was described as a rusty-mottle-like disease of sweet cherry (*189*) in recognition of the fact that the similarities of the diseases in the two States were not as clear-cut as formerly assumed. Subsequent studies of the various types of rusty mottle disease expressions throughout Utah and in the Northwestern States indicate definitely that, even though the disease in Utah has many characteristics in common with the rusty mottle described by Reeves (*182*) and also with the mild rusty mottle reported by Zeller and Milbrath (*264*) in Oregon, there are certain critical symptomatological differences which justify considering the trouble in Utah a distinctively different disease. It is reported here for the first time under the name "necrotic rusty mottle." So far as known, necrotic rusty mottle of sweet cherry occurs only in Utah.

Economic Importance

Necrotic rusty mottle is a major problem in sweet cherry production in Utah. In a preliminary survey during 1944, 20.9 percent, or 27, of the 129 orchard blocks visited in 4 counties showed the disease (*200*). In 10 of the 27 affected orchards 10 percent or more of the trees were diseased. In 5 of these orchards 22 to 46 percent of the living trees were affected. Pronounced leaf necrosis, bud killing, devitalization, and frequent death of affected trees emphasize the seriousness of the disease.

Host Range and Varietal Susceptibility

Necrotic rusty mottle occurs principally on Bing, Lambert, and Napoleon (Royal Ann) varieties of sweet cherry (*Prunus avium*). The disease has been found occasionally on Windsor and once on an unidentified early black-fruited variety of sweet cherry. Black Tartarian is known to be a symptomless carrier of the necrotic rusty mottle virus. The disease has been transmitted to mazzard (*P. avium*) seedlings.

Symptoms

Trees affected with the necrotic rusty mottle virus show a delayed development of leaves and blossoms in the spring (pl. 16, *C*). Leaves on diseased trees appear healthy during the first 3 to 5 weeks after petal fall. After this period brown necrotic spots of varying size, shape, and distribution develop rather abruptly in the more mature leaves throughout the tree (pl. 17, *A*). The portion of the leaf blade that is necrotic is greater on some trees than on others. In mazzard seedlings and in the Lambert variety the initial necrosis may be so extensive that severe defoliation may result.

Two to three weeks after the onset of the initial symptoms many of the remaining leaves become prematurely senescent (pl. 17, *A*) and fall. This second partial defoliation reaches peak expression some 2 or 3 weeks before harvest. It may cause losses of 25 to 85 percent of the remaining leaves on the tree.

About the time the fruits ripen yellowish to rust-colored chlorotic areas appear in the older surviving leaves. These areas may be generally distributed, resembling closely the chlorotic area described for mild rusty mottle (*264*) and for the type of rusty mottle described by Reeves (*184*). Late in the season, usually after harvest, the necrotic areas in the remaining leaves frequently fall out, and produce a conspicuous shot-hole effect. These rusty chlorotic areas, the brown necrotic spots, and the shot holes constitute the most important diagnostic symptoms of the disease during the latter part of the season. Except for a comparatively few necrotic spots and rust-colored areas, the apical leaves on branches and water sprouts (suckers) seldom show any of the late-season symptoms.

Autumnal senescence of the leaves on diseased trees occurs 2 to 4 weeks earlier than that of leaves on healthy trees. It is characterized by prominent rings and line patterns of dark green on a background of yellow, brown, or brilliant red (fig. 43).

As the disease develops part of the buds and leaf spurs are killed. This killing results in bare, rangy branches with terminal tufts of foliage, as shown in plate 16, *B*. In the more advanced stages of the disease, the older branches

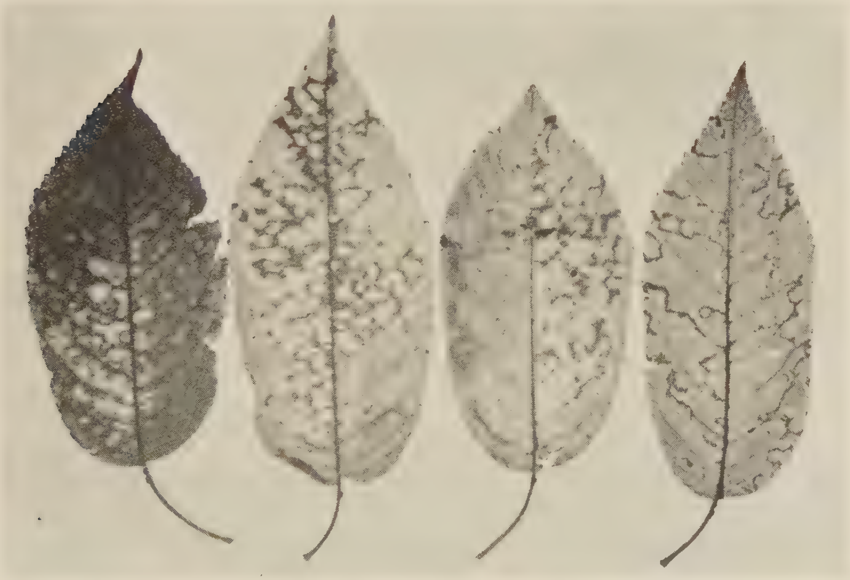


Figure 43.—Late-fall, senescent leaves from Lambert cherry tree inoculated with necrotic rusty mottle virus, showing rings and line patterns.

of the tree are killed and numerous water sprouts may develop from the trunk or from the lower portions of the main branches (pl. 16, *B*). On the cortex of young branches of some cherry varieties there occur numerous cankers, or blisterlike lesions, which as growth continues cause pronounced roughening of the bark. Frequently this feature is so prominent that it provides a very significant symptom of the disease.

Transmission

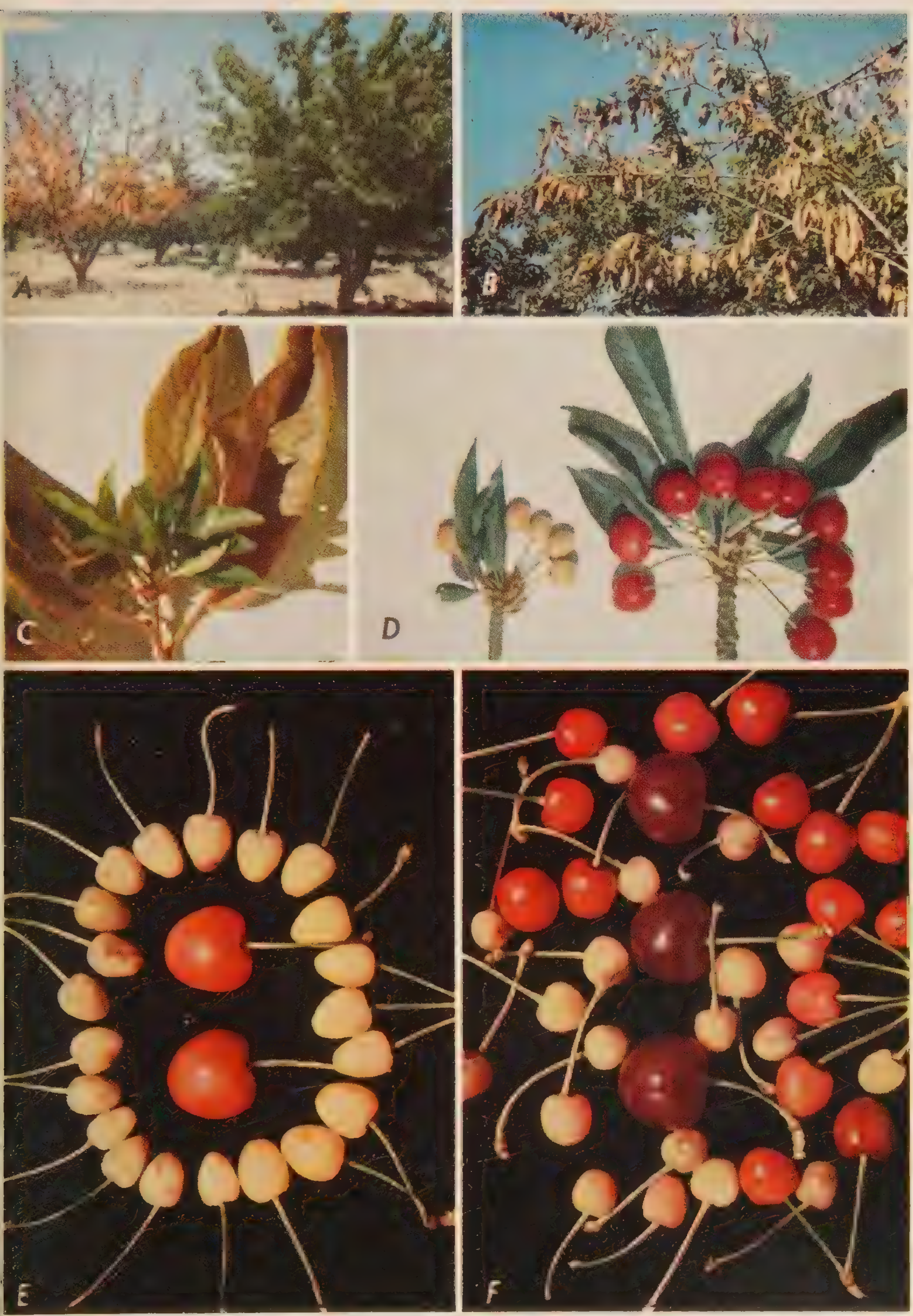
The virus of necrotic rusty mottle has been transmitted by budding with equal facility to mazzard seedlings and Bing, Lambert, and Napoleon varieties of sweet cherry. Transmissibility by this method is high, approaching 100 percent. Peach (*P. persica*), western chokecherry (*P. virginiana* var. *demissa*), Italian Prune (*P. domestica*), and certain varieties of sour cherry (*P. cerasus*) failed to produce visible symptoms when grafted with buds from diseased sweet cherry trees.

Incubation Period

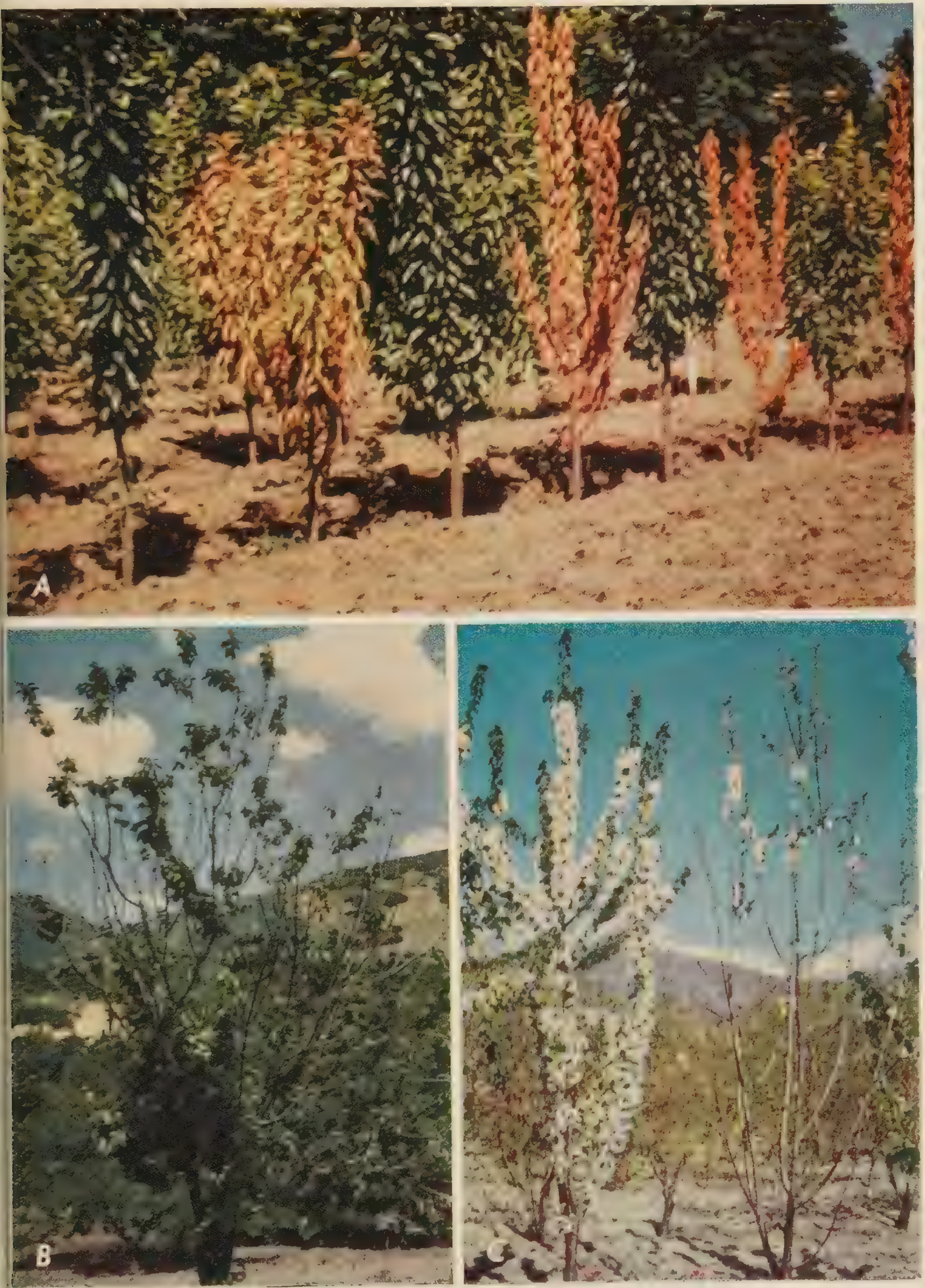
Trees inoculated in late August and early September exhibit symptoms the following spring.

Control Measures

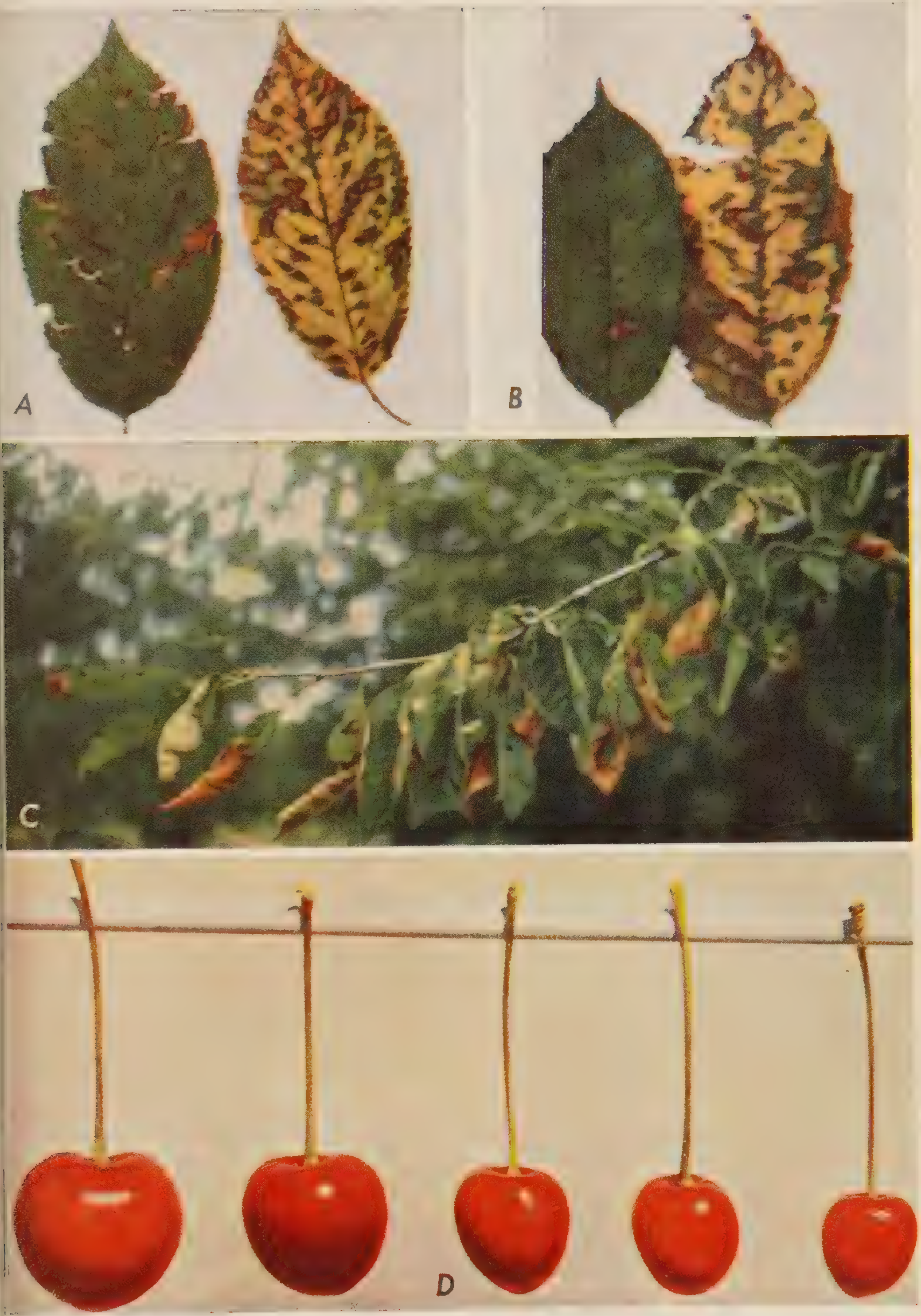
Diseased nursery stock provides the most common means of distribution of necrotic rusty mottle, but there is some evidence of natural spread in orchards. The use of virus-free budwood, both for propagation and for establishing pollinizers in orchards, offers the most effective method of control. Late-season inspection and roguing of nursery stock just before leaf fall or before frost in the fall may prove to be an additional safeguard in preventing the distribution of the disease in nursery stock.



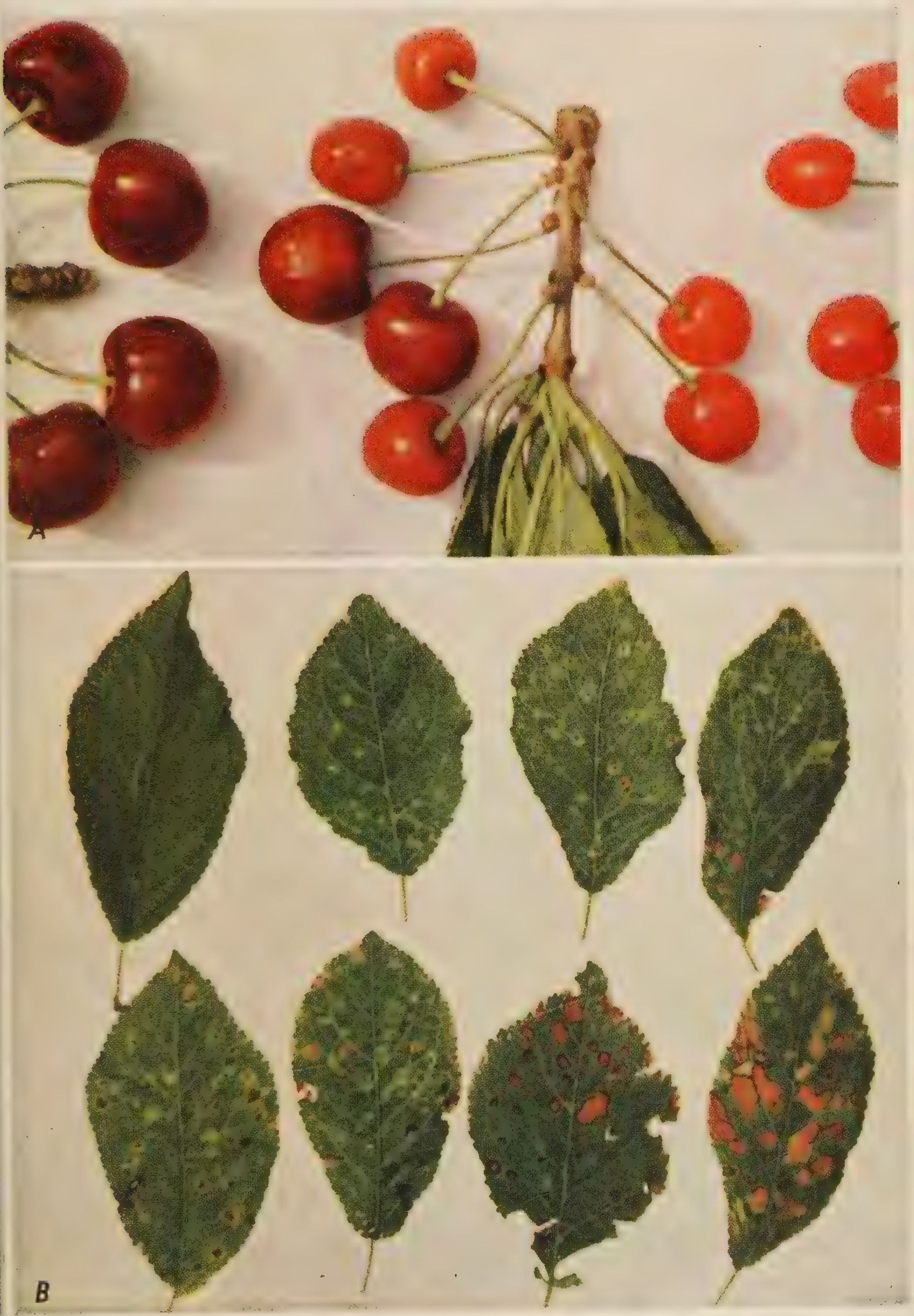
A, Dying albino-affected sweet cherry tree, contrasting with nearby normal trees. *B*, Branch of albino-affected sweet cherry tree, showing late-summer symptoms. *C*, Terminal from a similar tree, showing small green and older greenish-bronze leaves. *D–F*, Sweet cherry fruits affected with albino, contrasting with normal fruits: *D*, Lambert; *E*, Napoleon; *F*, Bing.



A, Bing cherry trees on mahaleb rootstock, showing dying of alternate trees, which were inoculated with albino virus the previous fall; *B*, 14-year-old Lambert cherry tree showing bare, rangy limbs and tufted terminals characteristic of advanced stage of necrotic rusty mottle; *C*, young Lambert tree inoculated with buds from tree in *B*, showing retarded leafing and blossoming.

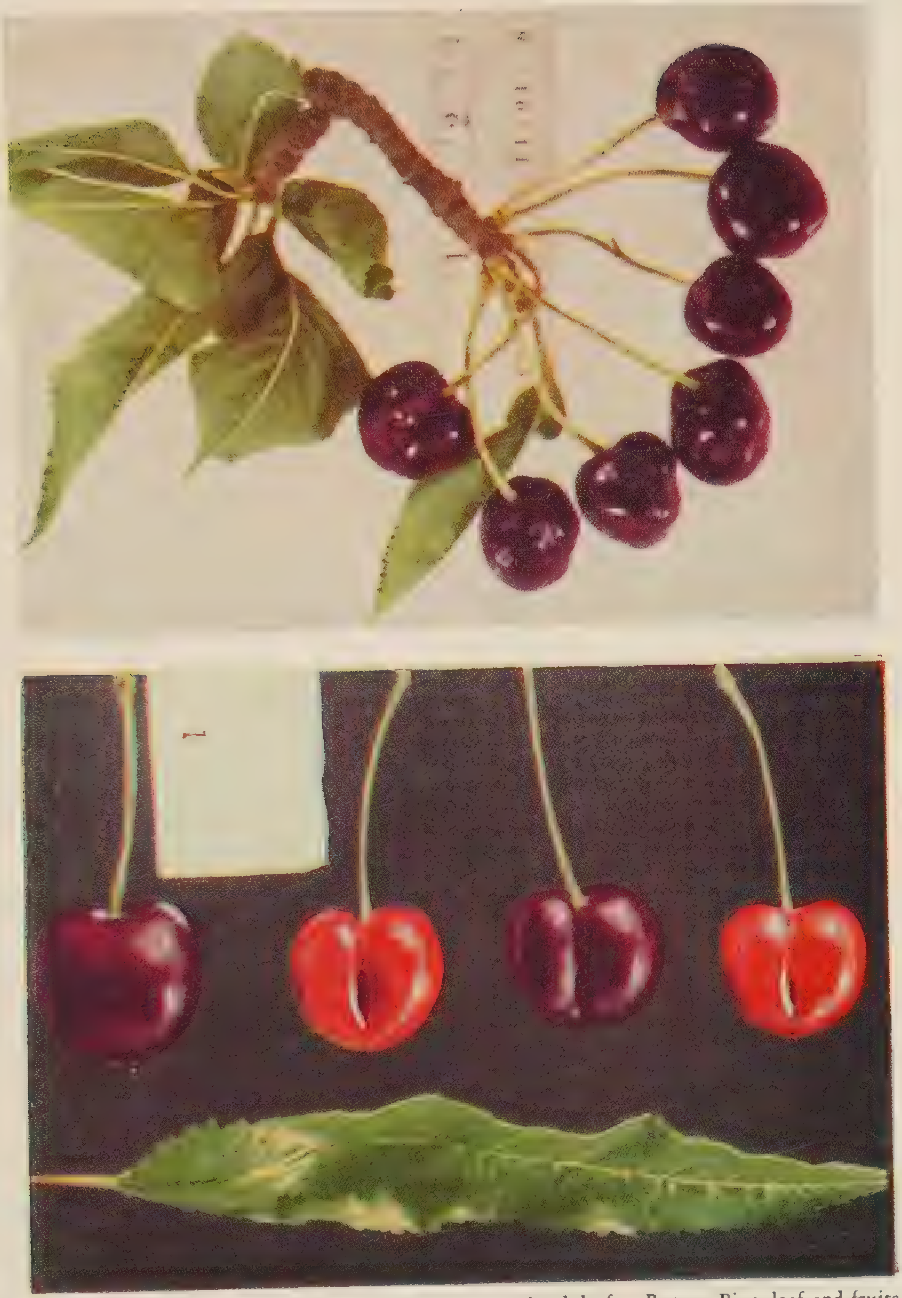


A, Lambert cherry leaves affected with necrotic rusty mottle, showing early stage (left) and senescent stage (right); *B*, Lambert cherry leaves affected with Lambert mottle, showing early stage (left) and senescent stage (right); *C*, shoot from Lambert cherry tree affected with an apparently virulent form of Lambert mottle; *D*, Lambert cherry fruits affected with little cherry, showing gradient in size as contrasted with normal fruit (left).

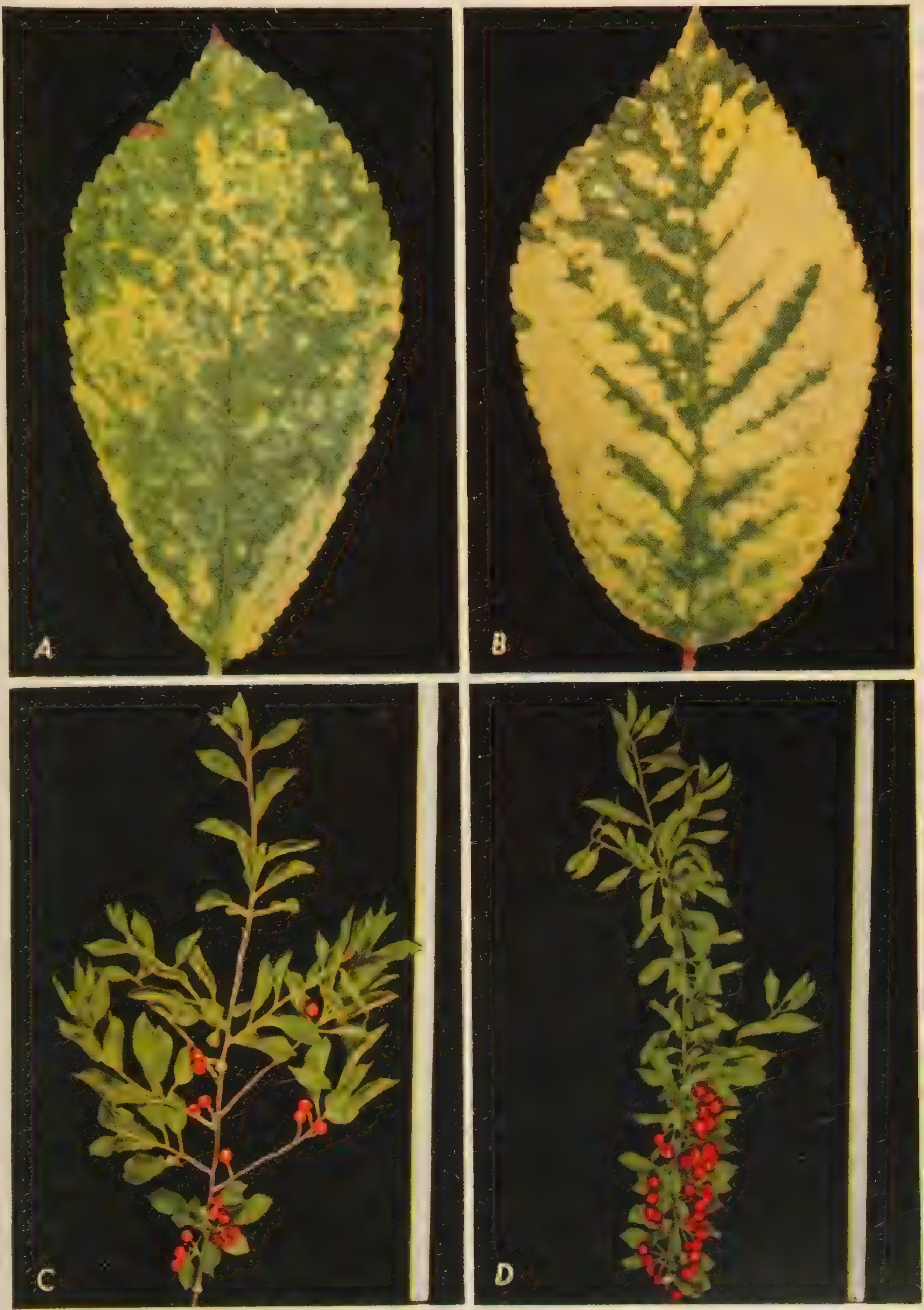


A, Bing cherry normal fruits (left) and spur from tree affected with small bitter cherry, showing two normal fruits and five affected ones; *B*, Italian Prune leaves arranged to show a gradient of Italian Prune leaf spot symptoms, in contrast with normal leaf (upper left).

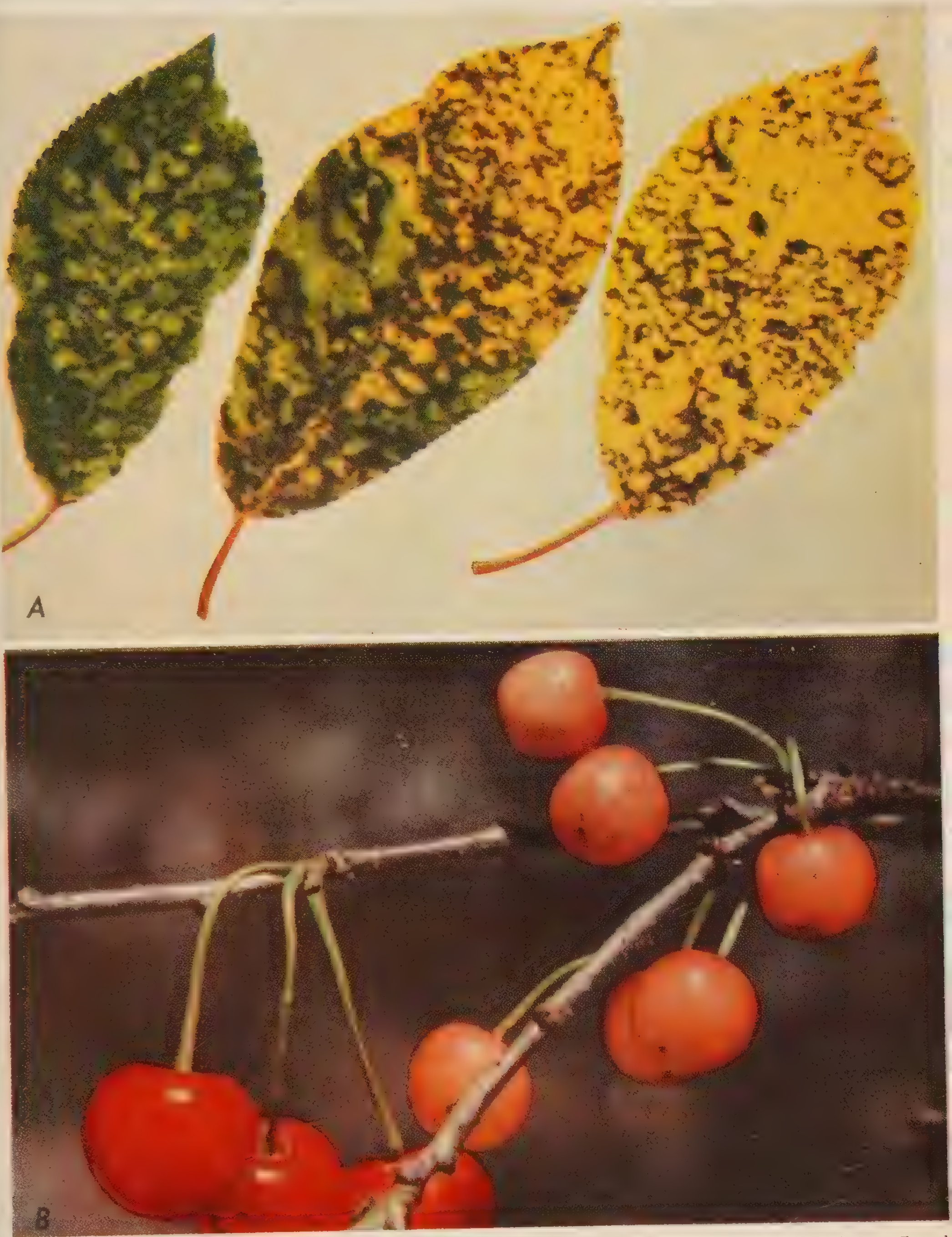
PLATE 19



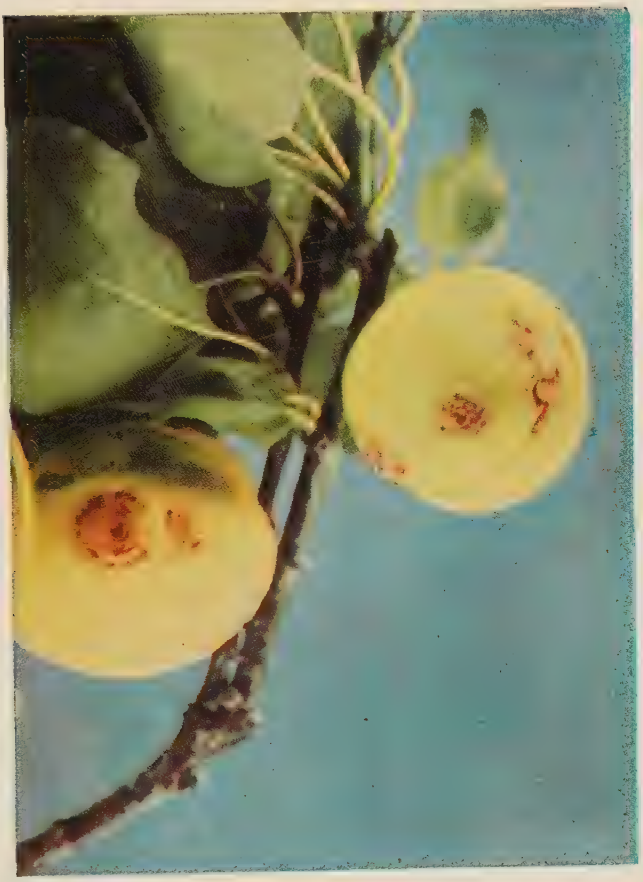
Top: Bing cherry leaves and fruits affected with twisted leaf. *Bottom:* Bing leaf and fruits affected with sweet cherry deep suture, in contrast with one normal fruit at left.



A–C, Sour cherry yellows on Montmorency cherry: *A*, Leaf showing irregular pale-green or yellowish mottling; *B*, leaf showing retention of green along main veins; *C*, branch showing willowy type of growth. *D*, Comparable normal branch with fruit spurs.



A, Montmorency cherry leaves showing symptoms of green ring mottle; *B*, Montmorency cherry fruits affected with pink fruit, in contrast with normal fruits.



Wenatchee Moorpark apricot showing symptoms of ring pox on the fruit.

LAMBERT MOTTLE

By T. B. Lott

Name of the Causal Virus

Lambert mottle virus.

Geographic Distribution

Scattered affected sweet cherry trees occur in various parts of the Okanagan Valley, British Columbia, Canada. The disease (*141*) has not been observed in the Kootenay area of British Columbia, but was transmitted by Napoleon (Royal Ann) cherry budwood obtained in that area.

Economic Importance

In most orchards the percentage of affected trees is low, though individual trees may be seriously affected.

Host Range and Varietal Susceptibility

Sweet cherry (*Prunus avium*) variety Lambert is affected, and symptoms develop on it. The Bing variety is infected, but there is little or no symptom expression. The Napoleon variety is probably a symptomless carrier.

Symptoms

The terminal shoots of Lambert trees on which the disease is well established appear normal in the early spring; but as the season advances, all the buds on the upper portion of many of these shoots either fail to open or swell and

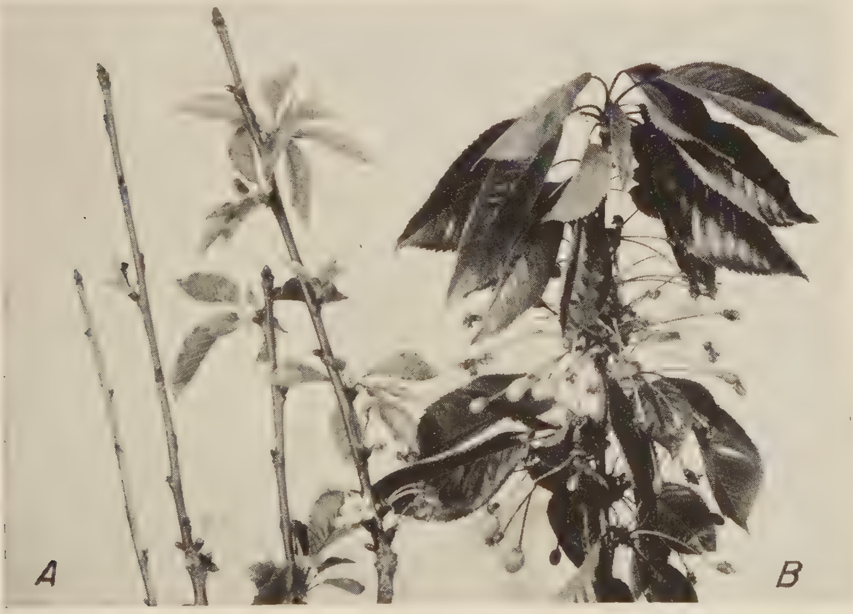


Figure 44.—*A*, Shoots of Lambert cherry affected with Lambert mottle, showing death of upper buds and delayed and irregular development of flowers; *B*, comparable healthy shoot.

ultimately die (fig. 44). The development of the other leaf buds and of the flower buds is both late and irregular. The one symptom which can be observed at all times is the form of branching that results from the production of new shoots part way down the previous season's growth.

In late spring the foliage appears slightly thin, but individual leaves are normal in appearance and most of them are full-sized. In early June a yellowish interveinal mottle begins to appear on the older leaves (pl. 17, *B*). This is soon followed by numerous small spots of a purplish or chocolate color, which later becomes brownish. These spots form lines alongside the veins and also irregular lines and rings or partial rings without relation to the veins. The lines of minute purplish spots are usually surrounded by a poorly defined greenish-yellow border. In some leaves a similar greenish-yellow pattern occurs without any purple spots.

In addition to these symptoms, and occurring without apparent relation to them, there are areas of the leaf up to 3 cm. in length which become brown and torn, but do not usually separate cleanly at the margin. Typically these spots are irregular in outline, but sometimes they extend as a narrow line along a vein. The brown areas occur on any part of the leaf blade except the midrib.

In midsummer the normal green of the older affected leaves changes to yellow and the greenish-yellow pattern becomes slightly more green. At this time defoliation of these leaves commences. The amount of defoliation varies from year to year. Half of the leaves may fall prematurely.

Diseased trees set only a light crop, and sometimes many of the fruits do not reach maturity. In some cases fruits of normal size and color have abnormally short and curved pedicels. On some trees nearly all the fruits arise from single buds on the lower part of the 1-year-old wood or a few surviving spurs on the outer part of the 2-year-old wood. The number of fruit spurs may be very greatly reduced.

The disease becomes progressively more serious for several years, and twigs and larger branches die. Trees affected when young may die, but in older trees the disease appears to become stabilized.

Diagnostic or Unusual Characteristics

The type of branching that results from the death of successive terminal buds and the subsequent growth from lateral buds is characteristic.

Transmission

The disease is transmitted without fail to Lambert trees by budding or grafting when the diseased inoculum makes union. It has also been transmitted by cambium scrapings placed under the bark.[23]

Incubation Period

Leaf symptoms appear the first year after bud inoculation. Twig symptoms do not appear until the second or third year.

Control Measures

Since Lambert mottle does not appear to spread rapidly in the orchard, it is likely that it can be eliminated by removal of affected trees. Care should be taken to use healthy budwood of all varieties for production of nursery stock.

Remarks

In recent years a disease which may be caused by a virulent and quick-acting strain of Lambert mottle virus has been observed in several places in the Okanagan Valley and also in one orchard in the Kootenay area. No transmission results are available. Trees affected with this disease have leaf markings similar to those caused by Lambert mottle. Leaf burning commences at the tip and margins and may affect most of the blade (pl. 17, *C*). Extensive twig, branch, and limb killing occurs, and within a few years a tree is dead except for a few scattered twigs and water sprouts which arise from the bases of the limbs. In one 5-acre orchard nearly all the trees of the Lambert variety were rendered worthless in about 10 years, but trees of several other varieties showed no visible effect.

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[23] McLarty, H. R. Unpublished data.

Symptoms of the above-mentioned disease closely resemble those of a disease in Utah (*189*) now referred to as necrotic rusty mottle, according to Reeves.[24]

Reeves reported observing similar symptoms on sweet cherry trees in three orchards near Wenatchee, Wash. He indicated that the disease in Washington and that in the Kelowna district of the Okanagan Valley are similar in their effect on the tree, particularly in regard to the necrotic leaf areas and general foliage symptoms; the extensive twig, branch, and limb killing; and the roughened bark that develops on many branches.

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[24] Reeves, E. L. Correspondence.

LITTLE CHERRY

By W. R. Foster, T. B. Lott, and M. F. Welsh

Name of the Causal Virus

Little cherry virus.

Geographic Distribution [25]

Little cherry was first observed in 1933 in a single orchard near Nelson, British Columbia, Canada. It is now widely distributed in the scattered cherry plantings in the mountainous southeastern part of the Province, along Arrow Lakes and to the east of them. It has not yet been found in the Okanagan Valley, where the main cherry plantings occur. A little cherry disease has also been reported in Washington (*185*), Idaho (*168*), Oregon (*265*), and Utah (*198*), but its relationship to the one in British Columbia is not known.

Economic Importance and Natural Spread

Little cherry is the most important virus disease of sweet cherry in British Columbia, because it reduces the value of the crop and its natural spread is extraordinarily rapid. When the disease becomes established some cherries are unmarketable. The majority can be processed into crystalized fruit, which is likely to be a somewhat inferior product. Some cherries can be marketed as fresh fruit, but they lack the flavor, sweetness, and attractive appearance of normal cherries.

The rate of natural spread of little cherry is phenomenal. Usually all the trees in an orchard are affected 2 or 3 years after the disease is first observed. Information received from several sources (69) indicates that the disease first appeared in 1 orchard in 1933. In 1934, 3 nearby orchards were affected. By 1940 the disease had spread in an area about 20 miles in diameter. In 1947 it was found in all the fruit-growing districts of the Kootenays, an area about a hundred miles in diameter. This rapid spread took place in spite of many natural barriers such as high mountains and large lakes and often considerable distances between orchards, in one case about 30 miles. Few of the 30,000 cherry trees in the widely scattered orchards of the Kootenays are still healthy. No little cherry was detected in 1947, 1948, and 1949 in an annual inspection of the 68,000 cherry trees in the Okanagan Valley, just over a hundred miles west of the original infection.

Host Range and Varietal Susceptibility

Sweet cherry (*Prunus avium*) varieties Bing, Black Tartarian, Deacon, Lambert, Napoleon (Royal Ann), Black Republican, and Windsor are susceptible. Lambert and Black Republican are the most severely affected; Bing, Black Tartarian, Deacon, and Napoleon are moderately affected. Sour cherry (*P. cerasus*) has not been included in transmission tests but has been observed showing symptoms similar to those on sweet cherry. Other *Prunus* species are not known to be susceptible and have not been observed with suspicious symptoms.

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[25] In the present state of knowledge it is impossible to know how many different diseases or manifestations of the same disease are involved; therefore the description given here applies to the disease as it occurs close to the first-observed infection near Nelson, British Columbia.

Symptoms

The known symptoms on sweet cherry are confined to the fruits. Diseased trees set and retain large numbers of fruits. These apparently grow normally until the end of the green growing stage. Development is variously retarded from the time the fruit-ripening processes should commence. On diseased trees many cherries are still readily visible above the leaves at picking time, while on healthy trees the cherries are largely hidden under the leaves. Cherries may hang on diseased trees for months without ripening fully. At picking time many or all of the cherries on affected trees are half or less than half normal size and retain the light red of immature cherries (17, *D*). Even those cherries which are more nearly normal in size and color lack normal flavor and sweetness and are usually dull in appearance, without normal gloss.

There is some difference in symptom expression on different varieties. From year to year the severity of the disease varies a little in most varieties and noticeably so in the Bing. The fruits of the Lambert variety in the second and subsequent years of infection are rather uniformly affected, being about half normal size, dull red at picking time, angular, and pointed, usually with three flat sides tapering toward the distal end (fig. 45). On the Bing variety symptom expression is very variable. Usually the Bing is less affected than other varieties, but sometimes Bing trees have very small cherries almost lacking in color and dropping readily at about picking time. In mapped orchards a number of Bing trees which had definite symptoms of little cherry

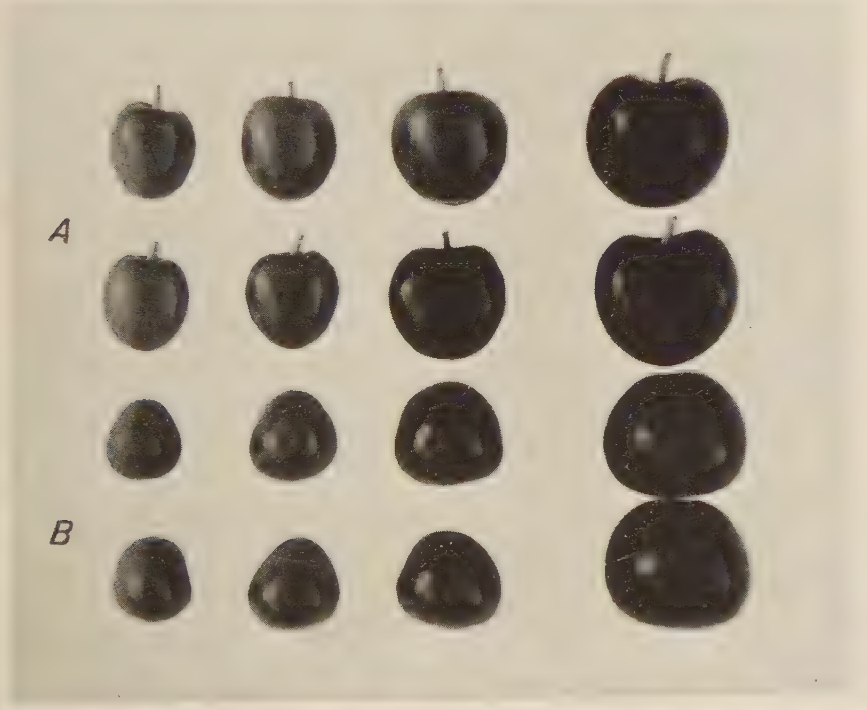

Figure 45.—**Lambert cherry fruits affected with little cherry, showing a gradient in size as contrasted with normal fruits at right: *A*, Side view; *B*, stylar-end view.**

for the first time in 1946 produced apparently normal fruits in 1947. There is a tendency for affected cherries of the Bing variety to be somewhat lumpy or irregular.

Symptoms of little cherry vary considerably in individual districts in the Kootenay area and often even in individual orchards in a given district. In certain orchards the effect of the disease on fruit size and color is very slight, even on the Lambert variety.

Diagnostic or Unusual Characteristics

The fruits are small in spite of the good foliage and the apparently healthy condition of the affected trees. Symptoms have been observed only on the fruits. Diagnosis is most certain close to normal picking time. Lack of flavor and sweetness even in fruits that appear normal is characteristic in all varieties. Pointed cherries with three flat sides tapering toward the distal end are typical in the Lambert variety. Natural spread is phenomenally rapid.

Transmission

Foster was the first to report definite transmission of little cherry in 1941, on 2 Lambert trees which had been budded with diseased buds in 1940 (*70, 73*). Check trees remained healthy. Foster and Lott (*73*) reported that 4 Black Republican (Republican) trees and 16 Lambert trees were budded in 1943 with buds from diseased trees. In 1944 all the Black Republican trees and 12 Lambert trees showed definite symptoms of little cherry, while 3 Lambert trees were highly suggestive of little cherry and 1 remained apparently normal. Check trees remained healthy. On most budded trees the part most distant from inserted buds was apparently normal at the time of the first crop after budding. Little cherry was evident throughout these trees the following year.

Welsh attempted to obtain the little cherry virus from untreated trees of Italian Prune (*P. domestica*), Vedette peach (*P. persica*), and two native species (*P. emarginata* var. *mollis* and *P. virginiana* var. *demissa*) by budding to healthy cherry. No definite transmission has been obtained. Attempts to transmit from cherry to these species and to Rochester peach and Moorpark apricot (*P. armeniaca*) also have given negative results. *P. emarginata* var. *mollis* budded in 1943 into a diseased Lambert tree produced apparently normal leaves and fruits up to 1949 even though the Lambert tree on which it was growing had been diseased for years.

Incubation Period

Trees bud-inoculated in the summer exhibit symptoms the following year.

Control Measures

The manner in which little cherry spreads is unknown. The rapidity of spread in an orchard and from one orchard to another is so great that an insect vector seems to be indicated. Prevention by tree removal is not certain to be successful in the absence of vector control. Control is not being attempted in the Kootenay area, where the disease is already prevalent. Control is being attempted, however, in the Okanagan Valley, where the disease has not yet been found. In 1947 there was begun a system under which the trees are inspected twice and, if any trees having little cherry or suspected of having it are found, they must be removed. The first inspection determines which trees are normal and which require further checking. The second inspection is made by those who are conversant with little cherry and other abnormalities which might be confused with it.

Remarks

No relationship is known to exist between little cherry and buckskin of sweet cherry or western X-disease of peach. Furthermore, in the Kootenay area of British Columbia the last has not been found in peach orchards, even though the peaches are growing adjacent to cherry plantings which have had little cherry for years. The little cherry that is present in the Kootenay area is distinct from small bitter cherry, which occurs in the Okanagan Valley always in proximity to peaches affected with western X-disease. A comparison of the symptoms of little cherry with those of small bitter cherry is given on page 130. The troubles known as western X little cherry in Washington, Idaho, Oregon, and Utah resemble in their fruit symptomatology the little cherry of the Kootenay area. However, no relationship with the Kootenay little cherry has been established.

The seriousness of little cherry is due to the severe reduction in quantity and quality of the fruit together with its unusually rapid natural spread. Experimental work has been rendered difficult by the inability of the investigators to detect the disease other than in the fruit.

SMALL BITTER CHERRY

By T. B. Lott

History and Geographic Distribution

Small bitter cherry has been present to a small extent in the southern part of the Okanagan Valley, British Columbia, Canada, since 1940 and probably somewhat longer.

Economic Importance

The effect of small bitter cherry is serious on some individual trees, but the disease is of negligible importance in the Okanagan Valley.

Host Range

Small bitter cherry was observed on only the Bing variety of sweet cherry (*Prunus avium*) before 1947. Three trees of the Lambert variety with symptoms apparently identical with those on Bing have now been found. Symptoms have not been observed on any other variety, and symptomless carriers are not known.

Symptoms

Small bitter cherry can be diagnosed most easily about normal picking time. Earlier diagnosis is not possible, and after picking time diagnosis becomes increasingly unreliable. Affected trees set about the usual number of cherries. These are of two kinds with few intergrades. Small cherries may be few, or they may constitute more than half the crop. Usually normal and small cherries occur together, but on some branches all the cherries may be normal and on others all may be small. It is common for only a small portion of a large tree to bear affected fruit. There may be little or no evidence of increased distribution of the disease in an affected tree for as long as 4 years. On the other hand, extensive increase in distribution may occur one year in a tree in which there was no increase in the preceding year.

On affected trees of the Bing variety the normal-appearing cherries are normal in size, shape, color, taste, and date of maturity. Most of the affected cherries are less than half normal size and are more oval than normal ones (pl. 18, *A*). At picking time they are bright red like immature cherries and somewhat bitter, with an objectionable flavor reminiscent of the smell of stagnant ditch water. They may hang on the tree for a month or more without ripening. After normal picking time they lose their bitterness and may taste somewhat fermented. On some trees the smallest fruits are pink or almost white and much underdeveloped at picking time.

Only three trees of the Lambert variety affected with small bitter cherry have been observed. The symptoms on the Lambert variety appear to be essentially the same as those on the Bing variety.

Diagnostic or Unusual Characteristics

Optimum conditions for diagnosis occur only within a few days of normal picking time. The main diagnostic problem is differentiation between small bitter cherry (*142*) and little cherry (*73*), so that little cherry can be recognized if it appears in the Okanagan Valley.

Trees of the Bing variety when affected with small bitter cherry produce fruits of two distinct types, some normal and others uniformly small, oval, and positively objectionable in flavor. Comparatively few fruits are either intermediate in size or exceedingly small. Trees of the Bing variety when affected with little cherry produce cherries with a more even gradient from the best to the worst. The best cherries are lacking in flavor and sweetness, and the worst are small, irregular in shape, and lacking in flavor and sweetness, but not positively objectionable in taste. Differentiation of these diseases is difficult and sometimes uncertain on trees of the Bing variety.

Differentiation of these diseases appears to be less difficult on trees of the Lambert variety. On Lambert trees affected with small bitter cherry, fruits are of two types: normal; and uniformly small, oval, light in color, somewhat bitter, and positively objectionable in taste. Lambert trees experimentally infected with the little cherry virus produced cherries that were uniformly undersized, flat-sided, triangular in outline when viewed from the distal end, dull in appearance, and lacking in flavor and sweetness, but not positively objectionable in taste.

Other differences between the diseases are that little cherry virus is easily transmitted and spreads rapidly within a tree whereas small bitter cherry virus moves slowly through a tree and the incubation period is long.

Transmission

Transmission of small bitter cherry has been obtained in only one tree, though eight trees were inoculated in 1941 and two in 1944. All these trees fruited after inoculation, and some are still under observation. Transmission was first obtained in 1949 in a tree that was inoculated in 1941. On this tree two branches have grown from scions from a diseased tree, and the third branch is part of the original inoculated tree. In 1947, 1948, and 1949 small bitter cherry was definitely present on the larger branch resulting from one of the inserted scions. Small cherries were few and scattered, and most of the cherries were normal. The smaller branch resulting from the other inserted scion has consistently produced only normal cherries. In 1949 small bitter cherry first became evident on the inoculated tree below the graft union, but most of the original inoculated tree continued to produce only normal cherries.

Control Measures

The removal of infected trees is recommended. Material for propagation purposes should originate from mature disease-free trees.

Remarks

Small bitter cherry has been found only in places where the western X-disease is fairly common on peach, but limited experimental work has produced no evidence of connection between the two diseases (*142a*).

RASP LEAF

By E. W. Bodine, Earle C. Blodgett, and T. B. Lott

Other Common Names of the Disease

Rasp leaf has been called leaf enation, ruffled leaf, and cockscomb.

Name of the Causal Virus

Rasp leaf virus.

History and Geographic Distribution

Rasp leaf was first observed in Colorado in 1935 by Bodine and Newton (*39*), and evidence of its virus nature was reported in 1942. Lott and McLarty (*96*) observed what is apparently the same disease in British Columbia, Canada, transmitted it, and called it leaf enation. Blodgett (*23*) reported its occurrence in Idaho and demonstrated its transmissibility. Reeves (*184*) in 1943 stated that rasp leaf had been observed in Washington for several years although not previously reported. Rasp leaf was reported to be present in Montana, Utah, and California (*184*).

Economic Importance

Observations in 1940 and 1941 indicated a rather rapid spread of the disease within orchards in Delta County, Colo., and many severely affected trees were found. In other districts where rasp leaf has been reported there is no evidence of rapid spread, and therefore in such districts it may be considered of minor importance. However, even in such districts (*23*, *184*) individual trees that are severely affected are markedly reduced in vigor.

Host Range and Varietal Susceptibility

So far rasp leaf has been reported on Bing, Black Tartarian, Lambert, and Napoleon (Royal Ann) varieties of sweet cherry (*Prunus avium*), on mazzard (*P. avium*) seedlings, and on Montmorency sour cherry (*P. cerasus*). The Napoleon variety appears to be the most severely affected. On some of the other varieties, and on mazzard trees especially, a milder manifestation of the disease is frequently encountered.

Symptoms

The most characteristic symptom of rasp leaf is the production of abnormal outgrowths on the lower surfaces of the leaves. These outgrowths (fig. 46) vary from elongated protuberances to leaflike growths. On a given leaf the growths usually occur between the veins and radiate from the midrib toward the margin of the leaf blade. On the upper surface of a diseased leaf there are depressed, roughened areas lighter in color than the normal green of the leaf. Since the majority of the growths on the lower surface of affected leaves resemble the teeth of a coarse rasp, the name "rasp leaf" was given to the disease. Severely affected leaves are small, narrow, and markedly distorted. The leaf blade frequently has a tendency to fold in upon itself. Trees may be completely or partially affected; it is not uncommon to find an uneven expression of symptoms on a branch or a tree. Damage to diseased trees consists of retardation in growth and the consequent reduction in the size of the crop.

Transmission

Transmission has been effected only through graft and bud inoculations. Trees inoculated in the fall may show symptoms during the following growing

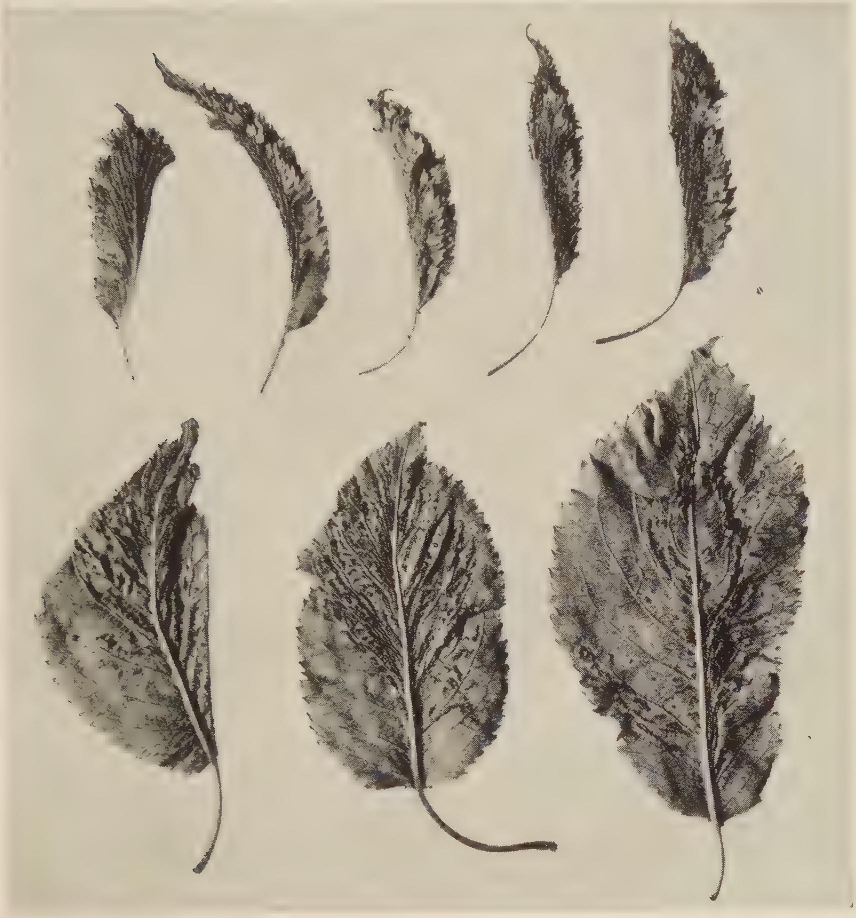

Figure 46.—Napoleon cherry leaves affected with rasp leaf, showing enations and distortions.

season, but the virus appears to be limited to parenchyma tissue in that it moves only a few inches from the inoculation point.

Incubation Period

The incubation period after inoculation may range from 9 months to 2 years.

Control Measures

Since the productiveness of severely affected trees is markedly reduced, it is recommended that such trees be removed. Furthermore, since the rasp leaf virus is transmitted by bud and graft inoculations, extreme care should be taken to obtain virus-free budwood for propagating nursery stock.

Remarks

Reeves (*184*) stated that it is not unusual to find casual leaf enations on cherry leaves scattered throughout healthy trees. This is true particularly of young trees and to some extent of older ones. The casual enations are not readily noticed unless the foliage is closely examined, whereas the leaf symptoms of rasp leaf are usually very striking in appearance. So far as known, these casual leaf enations are not of virus nature and therefore should not be confused with rasp leaf.

TWISTED LEAF

By T. B. LOTT and E. L. REEVES

Name of the Causal Virus

Twisted leaf virus.

Geographic Distribution

Twisted leaf occurs in the Okanagan Valley, British Columbia, Canada, and Chelan and Yakima Counties, Wash. A mild form of the disease, difficult to identify with certainty, probably occurs occasionally in Idaho and may occur in Oregon.

Economic Importance

Though twisted leaf does not have a wide distribution, it has caused considerable losses in a few orchards. According to surveys in Washington, reported by Coe (*66*), 147 infected trees out of a total of 199 were found in 1 orchard. However, in this orchard infection was largely due to the operations of the orchardist, who followed the practice of budding in pollinizer varieties. One of the pollinizers so employed was a mazzard seedling that exhibited only very meager symptoms and until indexed on Bing cherry could not be positively identified as affected with twisted leaf. Twisted leaf has seriously reduced production in 3 other orchards in Chelan and Yakima Counties, Wash., but insufficient information is available concerning the rate of spread of twisted leaf to indicate its threat to the section.

Host Range and Varietal Susceptibility

The Bing variety of sweet cherry (*Prunus avium*) is severely affected. Lambert, Napoleon (Royal Ann), Black Republican, and Black Tartarian varieties, mazzard (*P. avium*) seedlings, and an unidentified variety of sour cherry (*P. cerasus*) have been observed with very meager symptoms, but an insufficient number of affected trees of these have been discovered to permit conclusive statements regarding them. Limited experiments at Summerland, British Columbia, indicate that peach (*P. persica*) is also affected. A few Lambert and Napoleon trees remained symptomless after being budded with diseased buds.

Symptoms

Cherry.—Affected Bing sweet cherry trees are usually severely stunted, with shortening of the internodes particularly on the lateral branches. The spurs form compact clusters or tufts of leaves. Individual leaves are small and much distorted (pl. 19, *top*). Abrupt kinks occur in the midribs and petioles. Leaf blades are frequently bilaterally unequal. The severest distortion is accompanied by necrosis of parts of the midribs or lateral veins. Mottling may occur either as a peppering of small yellow spots with minute brown centers or more rarely as rather bright yellow lines close beside some of the lateral veins. Severe defoliation is sometimes observed (*140*). Reeves (*184*) reported severely misshapen fruits (pl. 19, *top*) on many trees in the Yakima Valley. Distinctive fruit symptoms have rarely been observed in the Okanagan Valley. Necrosis of the pedicel occurs when the fruit is misshapen. On cherry varieties other than Bing thus far observed, symptoms of twisted leaf have been either very meager or absent. With the information now available, identification of twisted leaf on cherry varieties other than Bing would be considered doubtful.

Peach.—At Summerland, British Columbia, twisted leaf has been transmitted from Bing sweet cherry to peach and back to Bing. The peaches showed a marked reduction in vigor and a cankering of the bark on the trunks and lower branches. Reeves has shown that peach trees inoculated with buds from some twisted-leaf-affected cherry trees produced no symptoms, yet when inoculating buds from other affected trees were used splitting and cankering of the bark resulted; the bark-cankering symptom is interpreted by Reeves as evidence that the cherry trees furnishing such inoculum were probably also carrying ring spot virus. Thus, the peach can be infected by the twisted leaf virus, but the symptoms if any have not yet been definitely determined.

Diagnostic or Unusual Characteristics

Twisted leaf can be clearly diagnosed on Bing cherry by the characteristically abruptly bent and twisted leaves. This effect is caused by necrosis and differential inhibition of normal growth of tissue in the petioles and main and lateral veins as compared with the leaf blade. Diagnosis on other varieties can be made by indexing on Bing.

Transmission

Transmission has been effected experimentally by budding and grafting. Preliminary-survey results indicate that spread usually takes place in the field at a slow rate. An exception was noted in one Yakima Valley orchard where a survey in 1946 showed a 15-percent increase of the disease during the preceding 3-year period.

Incubation Period

When inoculations were made on cherry early in the growing season, symptoms appeared within 2 months. When inoculations were made in the fall, symptoms appeared on the first growth formed in the spring.

Control Measures

Since the disease is known in only a relatively few orchards and there is little evidence of rapid orchard spread, diseased trees should be removed. Evidence indicating spread by grafting pollinizer arms of mazzard carrying the virus without recognizable symptoms onto Bing suggests the advisability of testing such pollinizer trees by indexing on Bing cherry before general use. Budwood sources in affected districts should similarly be tested lest the disease become widely disseminated in nursery stock.

Remarks

Severely and mildly affected Bing cherry trees have been observed in orchards. When transfers were made from such trees to Bing nursery trees, symptom expression agreed with that on the source material, indicating the presence of strains of the virus capable of producing different degrees of symptom expression. Serial transmission tended to reduce the severity of symptoms.

BLACK CANKER

By S. M. Zeller, J. R. Kienholz, and J. A. Milbrath

Name of the Causal Virus

Black canker virus.

History and Geographic Distribution

Black canker has been known in Oregon for about 25 years, but its nature was not determined until recently (*267*). It has been found in Hood River, Polk, Union, Wasco, and Washington Counties, Oreg. Reeves [26] reported "what appears to be the same disease" in Douglas and Yakima Counties, Wash. Lott [27] found a similar canker on a number of Napoleon (Royal Ann) trees in Okanagan Valley, British Columbia, Canada.

Economic Importance

Black canker is of little economic significance, because relatively few trees are affected. Most affected trees bear about as well as normal ones, but in certain instances black canker does considerable damage by causing severe dieback and thereby reducing fruiting wood.

Host Range

Black canker is known positively on only the Napoleon variety of sweet

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[26] Reeves, E. L. Unpublished data.

[27] Lott, T. B. Unpublished data.

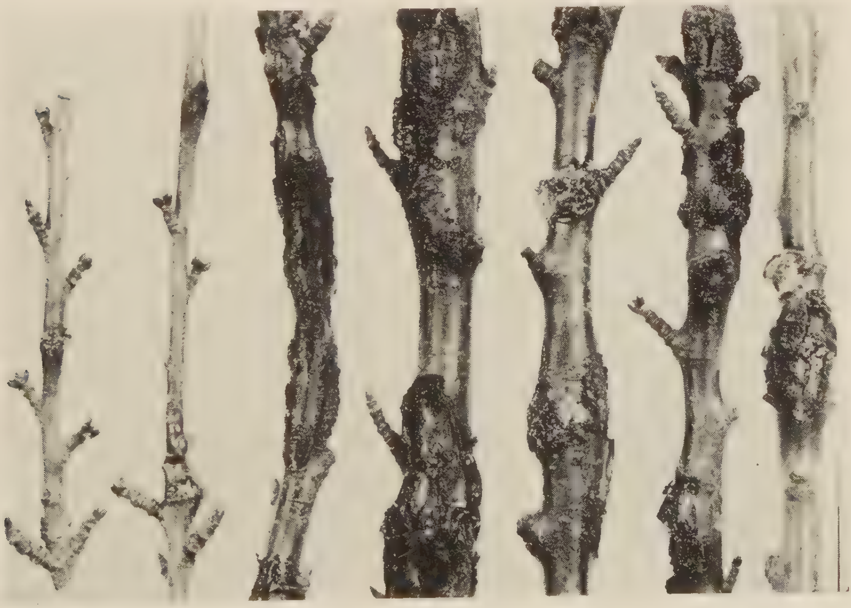

Figure 47.—Two- to four-year-old twigs of Napoleon cherry affected with black canker.

cherry (*Prunus avium*) in Oregon. The disease that Reeves [28] reported occurs also on the Bing, Black Republican, and Deacon varieties.

Symptoms

Cankers start on 1-year-old twigs, first as slightly swollen areas in which the bark splits lengthwise. These areas grow into rough black cankers, of which the ultimate size is more or less determined by the size of the affected branch (fig. 47). Some infected trees are very severely cankered, while others may have very few cankers. In severe cases dieback results indirectly from the cankers. No abnormal fruits and no leaf symptoms have been observed.

Transmission and Incubation Period

Transmission was brought about by graft inoculation, after a 2-year incubation period. No natural spread of the disease has been observed, but there is considerable evidence of spread through diseased propagation wood (buds or scions).

Control Measures

Budwood should be selected from uninfected trees.

Remarks

Many attempts to isolate a causal organism have failed.

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[28] See footnote 26, p. 137.

CHERRY RUGOSE MOSAIC

By H. Earl Thomas and T. E. Rawlins

Other Common Names of the Disease

Cherry mosaic 1.

Name of the Causal Virus

Festina lente Thomas.

Geographic Distribution

Occasional trees affected with cherry rugose mosaic have been seen in Alameda, Butte, Napa, Riverside, Santa Cruz, Solano, Sonoma, Sutter, and Tulare Counties, Calif.

Economic Importance

Observations begun in 1935 indicate that damage to infected trees is not great in any short period of years (*233*). The disease does not seem to be spreading readily in orchards.

Host Range

Natural infection is recognized on the Black Tartarian, Burbank, Napoleon (Royal Ann), Black Republican, and Rockport varieties of sweet cherry (*Prunus avium*). The virus was transmitted artificially to almond (*P. amygdalus*), peach (*P. persica*), and mahaleb cherry (*P. mahaleb*), and mild symptoms resulted.

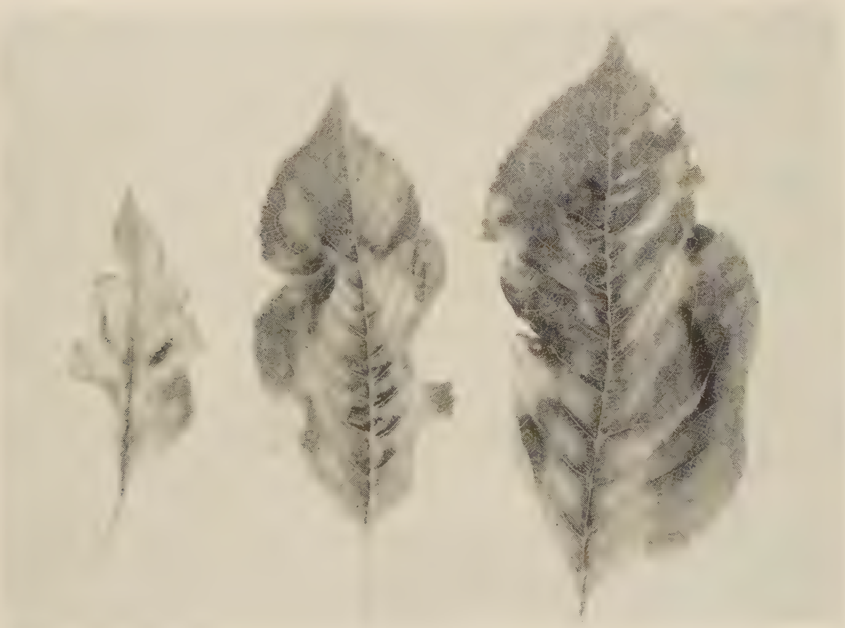

Figure 48.—Napoleon cherry leaves naturally affected with cherry rugose mosaic.

Symptoms

The most obvious symptom is in the leaf blade and consists primarily in a general chlorosis typically midway between the midvein and the leaf margin and accompanied by considerable distortion (fig. 48). Smaller lenticular chlorotic areas often appear along the lateral veins. Such affected leaves usually shrivel and drop by late June or July. The yield of fruit seems to be somewhat reduced, and certain individual fruits appear somewhat flattened on the suture side and more angular than normal.

Transmission

The disease is transmitted by grafting.

Incubation Period

Small Napoleon and mazzard trees inoculated by buds or scions from diseased trees in May and June developed symptoms early the following spring.

Control Measures

No specific measures have been undertaken to control cherry rugose mosaic. The use of buds from disease-free trees is advised.

TATTER LEAF

By R. S. Willison, G. H. Berkeley, and E. M. Hildebrand

Other Common Names of the Disease

The names "shredded leaf," "lace leaf," "ring spot," "necrotic ring spot," and "shot hole" have also been applied in various parts of the United States to similar, but not necessarily identical, viroses of sweet cherry.

Name of the Causal Virus

Tatter leaf virus.

Geographic Distribution

Tatter leaf has been found on sweet cherries on the Niagara Peninsula, Ontario, Canada, and in New York. A lace leaf condition has also been reported on sweet cherries in Washington (*183*) and Oregon (*256*).

Economic Importance

In Ontario tatter leaf is regarded as being of considerable importance on sweet cherry and of potential importance on sour cherry. Most of the susceptible sweet cherry varieties do not appear to be seriously affected by the disease, but field observations, supplemented by information from growers, indicate that the fruits of infected trees of certain varieties fail to ripen properly and that the general vigor of other varieties is impaired. In a survey conducted in Ontario in 1947 the incidence of tatter leaf was found to range from 0.6 to 41.3 percent in 24 of 26 sweet cherry orchards selected at random. In all, 2,649 trees were examined; of these 226, or 8.5 percent, were definitely affected with tatter leaf. An additional 371 trees, or 14 percent, showed mild symptoms suggestive of tatter leaf.

In New York (*95*) reduced vitality, accompanied by winter injury and reduction in yield and longevity, is associated with a tatter leaf condition in sweet cherry.

Host Range

The disease has been found in nature only on sweet cherry (*Prunus avium*). The known range of susceptible hosts comprises sweet cherry varieties Bing, Giant, Black Tartarian, Deacon, Elkhorn, Governor Wood, Lambert, Napoleon (Royal Ann), Schmidt, Seneca, Stark Gold, Windsor, Vernon, and Yellow Spanish and seedlings; sour cherry (*P. cerasus*) variety Montmorency; peach (*P. persica*) varieties Elberta and Rochester and seedlings; and mahaleb cherry (*P. mahaleb*) seedlings.

Transmission experiments indicate that domestica plum (*P. domestica*) varieties Italian Prune, German Prune, Lombard, and Reine Claude and Japanese plum (*P. salicina*) variety Abundance can also become infected, but symptoms are frequently masked.

Symptoms

Sweet cherry.—At the beginning of the growing season in Ontario, fine brown lines appear on many of the expanding leaves, outlining narrow interveinal areas, which later become necrotic, drop out, and give the typical lacy effect. Other leaves are mottled with yellow green or marked with oak-leaf and line patterns or rings (fig. 49). These symptoms usually persist throughout the summer but are found only on leaves formed early in the season. The

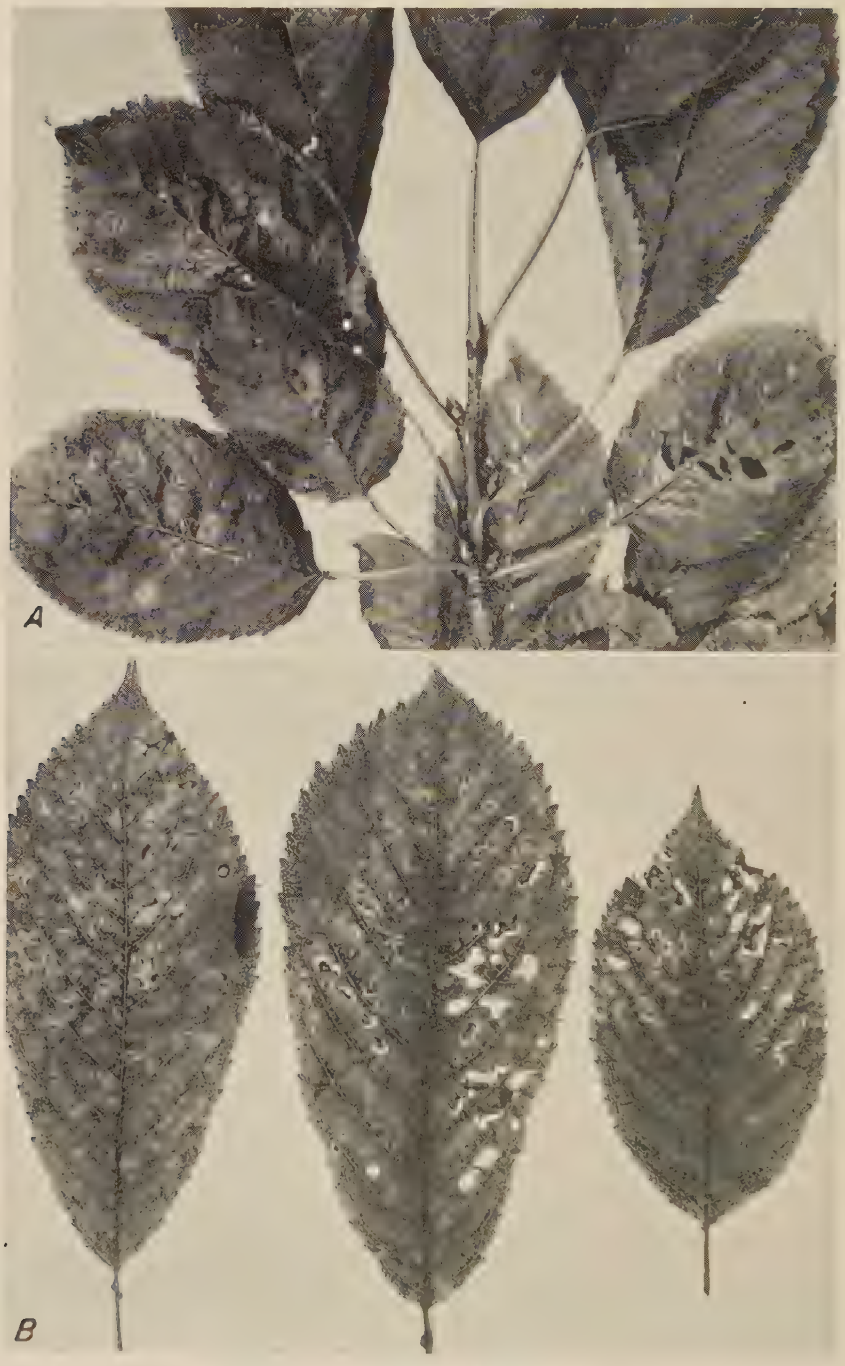

Figure 49.—*A*, Twig of Yellow Spanish cherry affected with tatter leaf; *B*, affected leaves of the same variety.

later leaves are normal in appearance and tend to hide the symptom-bearing leaves. Lacerations and patterns may reappear year after year, but in general there seems to be some reduction in the amount of symptom expression after the disease has become well established so that in time affected trees may sometimes become almost symptomless. Though observations on varietal susceptibility are not yet complete, some differences in the reaction of different varieties have been noted; for example, lacerations are less pronounced and less numerous on Napoleon than on Black Tartarian, while on Bing the necrosis often involves areas of the leaf surface larger than those just described and the affected leaves are distorted and rugose. On the variety Deacon an additional symptom is the failure of the fruits to ripen properly, while affected trees of varieties Elkhorn and Seneca usually show reduced vigor and dieback.

A second form of the disease also has been observed in one orchard on the Niagara Peninsula. Its symptoms differ from those already described in that the stages preceding the appearance of necrosis and laceration are characterized by red markings in place of the fine brown lines.

In New York severe terminal dieback was noted on Black Tartarian and Napoleon cherry trees within 6 weeks after inoculation by means of dormant diseased scions. Killing of young, vigorously growing Napoleon trees within 1 year was also recorded by Hildebrand.

Sour cherry.—In Ontario the first symptoms in the spring after artificial inoculation of Montmorency sour cherry resembled those of necrotic ring spot of sour cherry and consisted of fine translucent to dark-brownish, etched rings up to about 2 mm. in diameter and frequently confluent. There were also fawn-colored, red-rimmed necrotic spots of various shapes and sizes, which usually dropped out. The other markings either remained unchanged or gradually faded out. These symptoms, which were present only on the first few leaves to emerge, represent the initial, acute phase of infection.

In the second season there were some shiny lines and a trace of mottle and etched rings on a few of the first leaves. The foliage put out before the end of June was slightly rugose and undulant, but the later leaves developed normally and gradually obscured the early-season symptoms. In early July a few leaves turned yellow toward the base and dropped. A similar sequence occurred in the third year, but during the last 10 days of June there was considerable casting of yellow leaves marked with green pin spots. While the general effect was that of sour cherry yellows, the leaf markings were not quite characteristic of that disease.

In the third and subsequent years the leaves were shed 2 to 3 weeks earlier than usual in the fall.

The New York strains did not induce yellowing and premature dropping of Montmorency leaves except when mixed with a strain of the prune dwarf virus (*92*), but they caused severe terminal dieback of young trees inoculated by dormant grafting in the field. In the greenhouse Montmorency responded to some of these strains with terminal dieback and foliar symptoms very similar to those of necrotic ring spot of sour cherry.

Peach.—The initial, acute phase of the disease on peach was characterized by small green or yellow-green rings and by chlorotic and necrotic spotting of the first leaves to emerge the spring after inoculation. Slight superficial bark necrosis also occurred at this time, and more or less laceration of a few leaves appeared later in the season.

In later stages of infection the acute phase did not occur, but about the middle of the summer of the second and subsequent years scattered leaves throughout affected trees sometimes showed faint but definite mosaic markings, oak-leaf patterns, rings, or a premature aging of the upper leaf surface, which looks as if there were a very thin, finely checked, dull, dirty-yellow encrustation on it. On seedlings budded with tatter-leaf-affected material in the fall and cut back to the bud in the spring, the acute phase did not occur and the midseason symptoms appeared during the first season.

On peach, in New York, some strains of the virus induced terminal dieback and shortening of internodes, followed by recovery. Stem cankers appeared on peach after inoculation with a strain from a Yellow Spanish sweet cherry tree which had shown effects of winter injury only.

Peach trees inoculated with the Oregon strain sustained severe initial shock and showed lacy leaves scattered over the tree, dieback of branches, and large, sunken cankers around the inserted cherry buds (*256*).

Plum.—The plum varieties tested so far in Ontario and New York were little affected by tatter leaf; German Prune was symptomless, and Italian Prune, Lombard, and Reine Claude sometimes showed only very faint mottling or oak-leaf patterns. Scattered pinholes were observed in some of the midseason leaves of Abundance.

The virus strain associated with the second form occurring in Ontario induced symptoms on Italian Prune and peach that were indistinguishable from those associated with prune dwarf on these hosts, suggesting the presence of the prune dwarf virus in this bud source.

A New York strain which induced prune dwarf symptoms on Italian Prune (*92*) was shown to be much more heat-sensitive than the other strains.

Diagnostic or Unusual Characteristics

The laceration and chlorotic pattern on the first-formed cherry leaves in the early stages of infection are diagnostic of tatter leaf in Ontario, but in later stages the disease may sometimes be more or less masked. The disease is characterized by acute and chronic phases on most of the susceptible hosts.

Severe dieback, sometimes followed by death of young trees of certain varieties of sweet cherry, is regarded as characteristic of the disease in New York.

Transmission

Tatter leaf is easily transmissible by budding or grafting. No insect vector has yet been discovered, although considerable work has been done on this phase in New York.

Incubation Period

In outdoor experiments symptoms were not apparent on any of the hosts until the beginning of the next growing season after inoculation by budding or patch grafting even when the trees were inoculated as early as June. Two or more years may be required for the disease to become systemic in sweet cherry trees more than 2 years old.

Hildebrand found that in the field the incubation period on sweet cherry could be shortened to 1 month or less by grafting with dormant diseased scions just before growth starts. In the greenhouse symptoms appeared on rapidly growing seedlings of this species in 2 or 3 weeks after budding at different times during the growing season.

Control Measures

Roguing of diseased trees or seedlings is practicable in nursery rows, but not in orchards more than 2 years old. The use of indexed disease-free sources of scion and stock is recommended for propagation purposes.

Remarks

Although the symptoms induced on Montmorency sour cherry in the first year more or less resembled those of necrotic ring spot of sour cherry and in the third those of sour cherry yellows, the reactions of the differential hosts distinguish the tatter leaf virus with which Willison and Berkeley (*249*) worked from both of these sour cherry viruses as found in Ontario. Briefly, the separation is based on the following considerations.

In the first season the necrotic ring spot virus strains worked with in Ontario caused necrotic flecking on prune leaves and on peach a number of acute symptoms including delayed foliation and dieback, but they caused no annually recurring, chronic symptoms like those of tatter leaf. The symptoms induced on peach by return inoculations from Montmorency sour cherry artificially infected with tatter leaf were those described for tatter leaf, not for necrotic ring spot. Furthermore, in the Ontario trials the necrotic ring spot virus produced neither the typical tatter leaf, or lace leaf, conditions on sweet cherry nor the accompanying leaf markings; nor did it alone cause on Montmorency the sequence of symptoms described for tatter leaf.

It would also appear that the sour cherry yellows virus is not involved. The cherry yellows virus strains in Ontario and for that matter in eastern United States have all been found in combination with necrotic ring spot. The symptoms caused by combinations of sour cherry yellows and necrotic ring spot viruses also differ from those caused by tatter leaf virus. In the first place, these virus combinations induced internode shortening and persistent rosetting on peach, symptoms not associated with tatter leaf on peach in Ontario, as well as the acute symptoms characteristic of necrotic ring spot. In the second place, they did not cause typical tatter leaf markings and lacerations on sweet cherries.

In spite of the resemblance of the respective symptoms on sweet cherry, the tatter leaf condition found in New York was obviously caused either by a virus, or a strain of virus, differing in many important details from that found in Ontario or by a mixture of viruses. It is agreed that the differential-host reactions obtained by Hildebrand with the New York strains were very similar to, if not identical with, those obtained with the virus or virus complex causing necrotic ring spot of sour cherries. The reaction of peach to lace leaf of sweet cherry in Oregon (*256*) suggests affinity between that virus and the ring spot complex. Cochran and Hutchins (*62*) also obtained lace leaf symptoms on sweet cherry by inoculating with ring spot virus of peach. It seems probable therefore that at least two viruses are involved; their identities cannot always be readily distinguished by inspection of their symptoms either on sour or on sweet cherry, but they can be separated by differences in their effects on a range of differential hosts.

Furthermore, the possibility that the tatter leaf virus can be contaminated with other viruses such as necrotic ring spot and prune dwarf or vice versa should not be overlooked. From the practical point of view it may not matter much which virus is present, since neither virosis is desirable in an orchard or nursery, but their separation and recognition is important to the fundamental knowledge of the highly complex cherry virus problem. However, much remains to be done—both in continuing the differential-host-range studies and in supplementing them by other methods—before complete clarification is reached.

Since prune dwarf virus strains may be carried more or less masked in sweet cherries (*92, 246*), the appearance of prune dwarf symptoms on Italian Prune or Lombard plum upon inoculation with the tatter leaf virus can be attributed to a mixture of tatter leaf and prune dwarf viruses in the affected sweet cherry under investigation.

PINTO LEAF

By J. R. Kienholz

Other Common Names of the Disease

None.

Name of the Causal Virus

Marmor pintofolium Kienholz (*126*).

History and Geographic Distribution

Pinto leaf was discovered at The Dalles, Oreg., in June 1943 on Napoleon (Royal Ann) cherry. Later it was found at Hood River, Oreg., on mazzard seedlings. It is known definitely at these two localities only, but there is an unconfirmed report that it was seen at Kennewick, Wash.

Economic Importance

Pinto leaf is of very minor importance because of its limited occurrence. It is potentially dangerous if spread occurs, the degree of injury being about the same as that from mottle leaf.

Host Range

The disease has been found occurring naturally in the field on sweet cherry (*Prunus avium*) varieties Napoleon, Black Republican, and Stark Gold and on mazzard (*P. avium*) seedlings. Attempts have been made to transmit the trouble to sour cherry (*P. cerasus*), peach (*P. persica*), domestica plum (*P.*

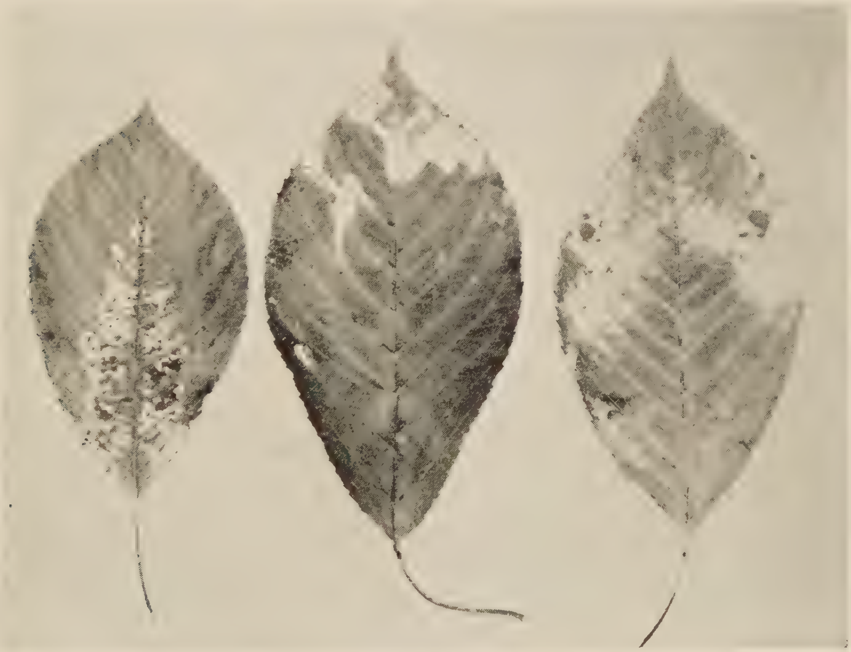

Figure 50.—**Napoleon cherry leaves affected with pinto leaf.**

domestica), apricot (*P. armeniaca*), western chokecherry (*P. virginiana* var. *demissa*), rose (*Rosa* sp.), apple (*Malus sylvestris*), and pear (*Pyrus communis*); but the difficulty of budding in the dry Dalles district makes these tests somewhat inconclusive.

Symptoms

Pale-green or yellowish patches on the leaves (fig. 50) soon change to bright yellow or white. No specific pattern is formed, but the small to large patches occur on any part of the leaf. Leaf symptoms are sometimes meager or obscure, particularly on mazzard seedlings, on which they often appear merely as a coarse stippling. Leaves on terminal shoots are rarely affected until late in the season when a few of the basal leaves may show the symptoms. A slight stunting of the trees occurs after several years, presumably because of the reduction of chlorophyll in the leaves.

The fruits never quite reach full maturity in color or taste, being rather insipid, like the fruits from trees affected with mottle leaf.

Transmission

So far pinto leaf has been transmitted by budding only.

Incubation Period

Trees bud-inoculated during the early fall show leaf symptoms early the following spring.

Control Measures

At present no control measures have been tried.

ROUGH BARK OF ORIENTAL FLOWERING CHERRY

By J. A. MILBRATH and S. M. ZELLER

Names of the Causal Virus

In accordance with Holmes' system of classification the name "*Rimocortius kwanzani* Milbrath and Zeller" was applied to the virus and "Prunus virus 9 Milbrath and Zeller" was assigned in accordance with Smith's system (*155*).

Geographic Distribution

The disease has been reported from Oregon only.

Economic Importance

Serious losses were sustained in nurseries from poor bud set and the production of misshapen, dwarfed trees until rough bark was eliminated from the stock. Affected trees do not recover and are therefore a complete loss.

Host Range and Varietal Susceptibility

Only the Kwanzan variety of oriental flowering cherry (*Prunus serrulata*) is known to be naturally affected. Shirotae (Mount Fuji), Naden, and Shirofugen varieties have been artificially infected. Mazzard (*P. avium*) seedlings, the Bing, Napoleon (Royal Ann), and Lambert varieties of sweet cherry (*P. avium*), and the J. H. Hale peach (*P. persica*), all of which also have been artificially infected, retain the rough bark virus without symptom expression. Normal vigorous shoots of mazzard often grow from the base of infected Kwanzan trees. Such mazzard understock retains the virus.

Symptoms

Nursery trees of oriental flowering cherry variety Kwanzan propagated from infected stock are dwarfed by extreme shortening of the internodes, which makes the leaves close together in clusters. Areas on the midribs of the leaves become necrotic and crack, causing the leaves to arch downward (fig. 51, *A*). The dwarfing of the growth and the curving of the leaves cause an effect similar to that caused by aphid injury. The bark of current-season growth becomes deep brown and roughened by longitudinal splitting (fig. 51, *B*). This splitting often originates at lenticels. On wood 2 years old or older this splitting and the rough bark condition become more pronounced and new growth continues to be much reduced. Shiro-fugen develops much the same symptoms as Kwanzan except that the bark splitting is not so evident. Naden develops only a slight cupping of some of the leaves as a result of necrosis and splitting of the midrib and main lateral veins. The growth is not dwarfed, and no bark splitting occurs. Shirotae does not show any definite symptoms.

Transmission

The virus is readily transmitted by budding or grafting. Complete organic union is not necessary, and in many cases the virus is transmitted even when the bud dies shortly after budding. Many mazzard seedlings budded in the nursery were infected even when there was no apparent union between the Kwanzan bud shield and the mazzard rootstock.

Incubation Period

Kwanzan bud-inoculated in August or September shows symptoms throughout the tree as soon as new growth develops in the spring.

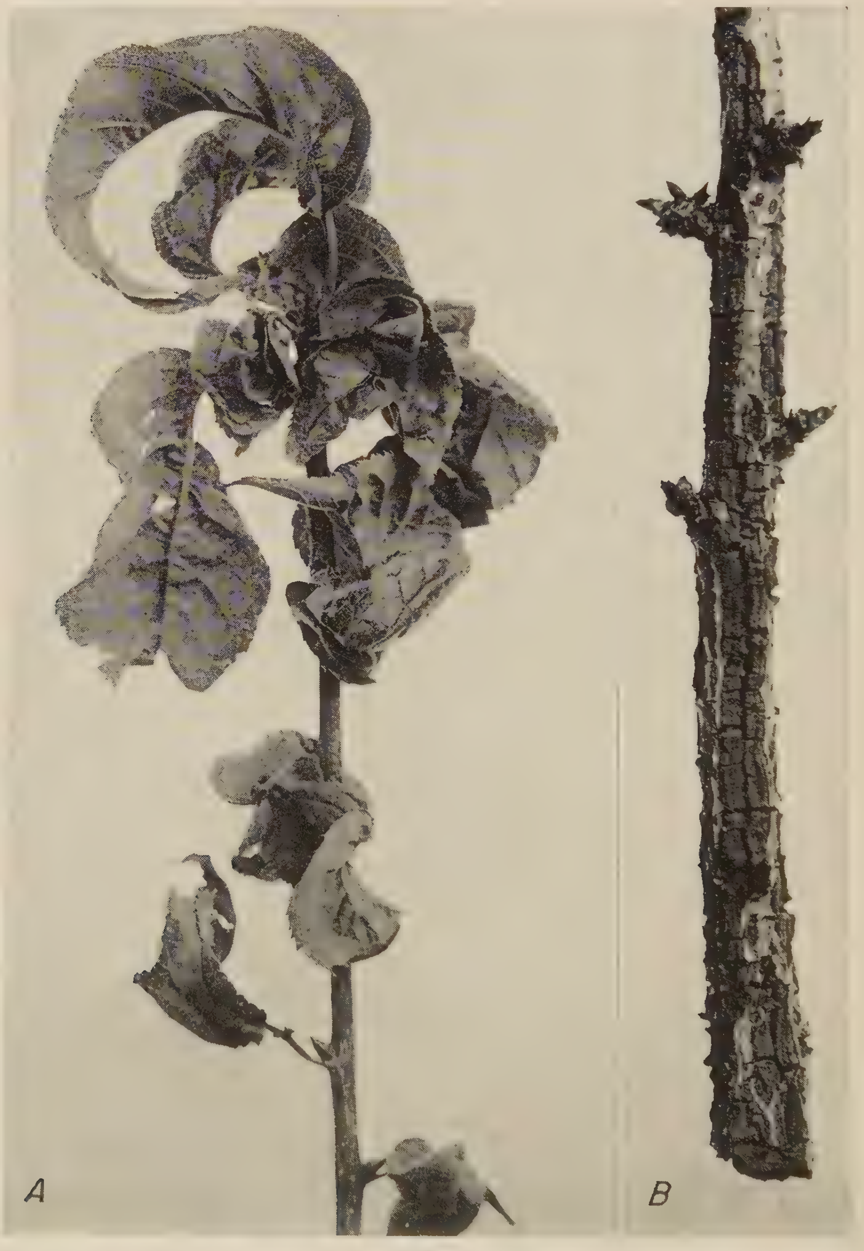

Figure 51.—*A*, Current-season branch of Kwanzan oriental flowering cherry affected with rough bark, showing characteristic cupping and arching of leaves; *B*, older branch showing severe splitting and rough bark caused by the virus.

Control Measures

The disease has been eliminated from the nurseries in Oregon by removing infected trees from the mother block and by using budwood from only Kwanzan trees showing good growth without rough bark.

VIRUS DISEASES OF SOUR CHERRY

SOUR CHERRY YELLOWS

By G. W. Keitt, G. H. Berkeley, Donald Cation, C. N. Clayton, E. M. Hildebrand, J. Duain Moore, and E. J. Rasmussen

Other Common Names of the Disease

Yellow leaf (*76, 177, 215*), physiological leaf drop (*68, 96*), physiological yellow leaf (*175*), physiological yellows (*177*), boarder tree (*124*) and virus leaf drop (*159*).

Names of the Causal Virus

The common name is "sour cherry yellows virus or viruses" (*124*). Hildebrand (*91*) proposed the name "*Chlorogenus cerasae.*"

Geographic Distribution

Sour cherry yellows or diseases with symptoms closely conforming to those of sour cherry yellows have been reported in the United States in California,[29] Idaho (*20*),[30] Indiana,[31] Iowa,[32] Michigan (*175, 176*), Missouri,[33] Montana,[34] New Hampshire,[35] New York (*68, 76, 98, 215*), North Carolina (*54*), Ohio,[36] Oregon,[37] Pennsylvania (*137, 138*), Utah,[38] Washington (*184*),[39] and Wisconsin (*121, 122, 123*) and in Canada in British Columbia [40] and Ontario (*8*). More or less sour cherry yellows has been found by Moore and Keitt to occur commonly in the sour cherry nursery stocks that they have indexed, though symptoms may not have been expressed in the nursery and the disease may not have been recorded in the States in which the nurseries were located.

Economic Importance

Yellows appears to be economically the most important known virus disease of sour cherry in the United States and Canada. It is widely distributed in the main areas of commercial sour cherry production in these countries. The percentage of trees affected varies greatly in different orchards. In some old orchards all or nearly all the trees are diseased. The rate of spread of the disease varies with the orchard and the season. Surveys in Wisconsin indicated annual increases in the percentage (based on total number of trees) of diseased trees per orchard commonly ranging from about 1 to 10 percent and averaging approximately 3 (*124*). Somewhat higher rates of spread were reported from Michigan by Rasmussen and Cation and from Ontario (*250*).

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[29] Wilson, E. E. Unpublished data.

[30] Blodgett, E. C. Unpublished data.

[31] Shay, J. R. Unpublished data.

[32] Buchholtz, W. F. Unpublished data.

[33] Millikan, D. F. Unpublished data.

[34] Morris, H. E. Unpublished data.

[35] Rasmussen, E. J. Unpublished data.

[36] Winter, H. F. Unpublished data.

[37] Zeller, S. M. Unpublished data.

[38] Richards, B. L., and Keitt, G. W. Unpublished data.

[39] Reeves, E. L., Johnson, F., Blodgett, E. C., and Keitt, G. W. Unpublished data.

[40] Foster, W. R. Unpublished data.

Yields from diseased trees fall off at variable rates and to variable extents. Commonly within 2 to 5 years after the first symptom expression the yield in Wisconsin may be reduced half or more (*160*). Trees that have been diseased for many years may show much greater reduction in yield. Attempts by Hildebrand (*90*), Rasmussen and Cation (*177*), and Moore and Keitt to restore the fruitfulness of trees affected with yellows have been unsuccessful.

Host Range

At present it is not possible to give an unqualified statement as to the host range or symptomatology of yellows. It is possible that more than one virus is concerned in the etiology and that each virus may have different strains. Numerous viruses and many strains may affect the species of *Prunus* used in the host-range studies. In some cases a species of *Prunus* inoculated with a virus, such as the necrotic ring spot virus of sour cherry, will show symptoms the first year and later carry the virus without showing further symptoms. Consequently, the facts that a host shows symptoms of virosis after inoculation with buds from a source of cherry yellows and that cherry yellows can be recovered from the intermediate host do not necessarily prove that the symptoms on the intermediate host were incited by the cherry yellows virus or virus complex. They may have been incited by one component of a cherry yellows virus complex or by some other virus or viruses carried in the scion, the stock, or both.

The following paragraphs are intended to present the available information, which is recognized as incomplete and in some aspects probably subject to reinterpretation in the light of future findings.

Symptoms characteristic of yellows are found in orchards and nurseries on sour cherry (*P. cerasus*) varieties Montmorency, Early Richmond, and English Morello. Yellows has been transmitted by budding or some other grafting method from Montmorency sour cherry to sour cherry varieties Montmorency, Early Richmond, and English Morello, and leaf symptoms characteristic of yellows have appeared. In greenhouse studies Moore and Keitt have transmitted cherry yellows back to Montmorency from all the cherry varieties just listed.

In Wisconsin Moore and Keitt have transmitted the virus or virus complex by budding from Montmorency sour cherry through each of the following hosts and back to Montmorency without the expression on these intermediate hosts of any recognized symptom characteristic of yellows on sour cherry: Sweet cherry (*P. avium*) varieties Bing, Napoleon (Royal Ann), and Black Tartarian and mazzard (*P. avium*) seedlings; peach (*P. persica*) varieties Elberta, Halehaven, and Rochester; domestica plum (*P. domestica*) varieties Bradshaw, Italian Prune, and Lombard; American plum (*P. americana*) seedlings; duke cherry variety Royal Duke; myrobalan plum (*P. cerasifera*) seedlings; mahaleb cherry (*P. mahaleb*) seedlings; black cherry (*P. serotina*) seedlings; eastern chokecherry (*P. virginiana*) seedlings; and pin cherry (*P. pensylvanica*) seedlings. Leaf symptoms different from those of yellows on sour cherry occurred on all these intermediate hosts, but they were not clearly distinguishable in these experiments from symptoms on the same hosts budded comparatively with buds from Montmorency affected with necrotic ring spot alone. They are, therefore, not attributed to yellows though it is recognized that the necrotic ring spot virus may be a part of a yellows virus complex and that further experiments may reveal differential symptoms on some of these hosts when they are budded comparatively with buds affected with yellows and with buds affected with necrotic ring spot but not with yellows. All attempts to transmit cherry yellows from Montmorency to Japanese plum (*P. salicina*) varieties Abundance and Burbank failed. No symptoms were produced on these two varieties, and yellows was not recovered from them by budding back to Montmorency (*163*).

In Ontario, New York, and Michigan the sour cherry yellows virus or virus complex was transmitted by grafting to peach varieties Elberta, J. H. Hale, South Haven, Halehaven, Kalhaven, Fertile Hale, and Rochester; to peach seedlings; to domestica plum varieties Italian Prune and Lombard; and to mahaleb cherry—by Berkeley, Hildebrand, and Rasmussen and Cation.

A fuller understanding of host relationships and symptoms must await further clarification of the relations of sour cherry yellows, necrotic ring spot, and possibly other virus diseases.

Symptoms

Sour cherry.—The most striking symptom on Montmorency sour cherry is yellow and green mottling of some of the leaves (pl. 20, *A*, *B*), followed by leaf casting. Chlorosis appears first as irregular pale-green or yellowish areas anywhere on the leaf lamina and may progress until the entire leaf is yellow. Some leaves may retain the green color longest along the larger veins (pl. 20, *B*). Leaves may be cast before any chlorosis is visible or at any stage of yellowing. The older leaves generally show symptoms first, often beginning with the scale leaves. When favorable conditions continue symptoms usually appear on the next younger leaves in the order of their age. If a wave of yellows is interrupted by a period of weather unfavorable for symptom expression, one or more leaves on a shoot may remain apparently healthy whereas leaves farther up the shoot may develop typical symptoms after favorable conditions resume. Most trees that have been diseased several years tend to have larger and fewer leaves than normal.

In the orchard the first wave of yellows is usually the most severe. In Door County, Wis., and in other areas with similar climates, it usually comes about 3 or 4 weeks after petal fall. Other waves of varying severity may occur until fall. The amount of defoliation due to yellows is variable, ranging from a trace to more than 50 percent of the leaves.

Trees of bearing age that have had yellows for several years generally show a much reduced spur system and a variable reduction in yield, averaging about 50 percent or more. The fruits average larger than normal and are of good quality.

Fewer leaf buds seem to be differentiated on the shorter terminal twigs on trees affected with yellows than on healthy trees. Furthermore, according to Moore and Keitt, leaf buds on yellows trees tend to grow into shoots (pl. 20, *C*). Thus there are more shoots, which tend to be somewhat shorter and thicker than those on healthy trees. The trees eventually develop a willowy type of growth (pl. 20, *C*), with long, bare spaces on the twigs, and the crop is produced largely on the shoots grown in the preceding season. Trees affected with yellows apparently may live about as long as healthy ones, but their trunks and branches tend to be more subject to breakage in windstorms (*124*).

The same symptoms were produced on Early Richmond sour cherry as on Montmorency (*124*).

The same leaf symptoms occurred on English Morello as on Montmorency. In Moore and Keitt's greenhouse work leaf symptoms did not appear until the third year after budding. In that year some English Morello fruits approaching maturity developed small necrotic lesions, usually internal, and became more or less distorted.

Peach.—In work conducted by Berkeley (*8*), Hildebrand (*84, 87*), and Rasmussen and Cation (*176*) with peach seedlings and varieties Elberta and Rochester, it was found that trees budded in the field in the fall with buds affected with the cherry yellows complex showed considerable necrosis, dieback, rosetting, and stunting of shoots. However, trees budded with buds affected with necrotic ring spot alone did not show rosetting, though necrosis and dieback were present. Moreover, such trees tended to recover, whereas when the cherry yellows virus was present the rosetting and stunting persisted from year to year. This differential reaction was reported to be clearly demonstrated when seedlings were cut back in the spring close to buds that had been inserted near the ground line in the fall. When cherry yellows virus was present, the shoots were dwarfed and rosetted (fig. 52). When cherry yellows virus was absent, the shoots were more or less normal, though some mottling might be present.

In Wisconsin greenhouse studies by Moore and Keitt with Elberta, Halehaven, and Rochester varieties of peach, inoculations at bud break with buds from two sources of cherry yellows incited rosetting only on Halehaven and only in the year of inoculation. Budding with material from two sources of necrotic ring spot alone did not incite rosetting on any of the three varieties. After two seasons in pots kept in the greenhouse except over summer, when they were out of doors, these trees were removed from the pots and planted in the field. There have been no rosette symptoms in the field, but yellows has been transmitted back to sour cherry from all three peach varieties budded originally with buds affected with yellows.

Plum.—In Ontario in the year after budding of Italian Prune and Lombard plums with buds affected with cherry yellows, small necrotic spots and some shot holes were present on a few first-formed leaves on the twig above the bud insertion. The following year definite prune dwarf symptoms were present on several branches. In this connection it was noted that, when Italian Prune was budded with buds affected with necrotic ring spot, small necrotic spotting also resulted on unfolding leaves, but prune dwarf symptoms were lacking. This suggested that the necrotic spotting was most likely due to the necrotic ring spot virus. In support of this hypothesis is the fact that in Berkeley's experiments buds infected with sour cherry yellows virus from two other sources known to contain the necrotic ring spot factor did not give rise to prune dwarf symptoms, but they did give rise to only the necrotic spotting referred to previously. Further data and possible explanations of the similarities and differences of symptom expression of cherry yellows and prune dwarf were given by Berkeley and Willison (*9*).

In greenhouse experiments by Moore and Keitt in Wisconsin, potted Italian Prune trees were inoculated by budding from the following virus sources: (1) Two Montmorency trees affected with necrotic ring spot, but not with yellows; (2) five Montmorency trees affected with sour cherry yellows and known to contain the necrotic ring spot virus that is latent on Montmorency after one season of symptom expression; and (3) one Montmorency tree affected with yellows and known to contain a strain of necrotic ring spot virus that incites striking recurrent symptoms on Montmorency annually. Each inoculated Italian Prune tree received two buds from a single tree of the given source, and four unbudded trees served as controls. All the budded prune

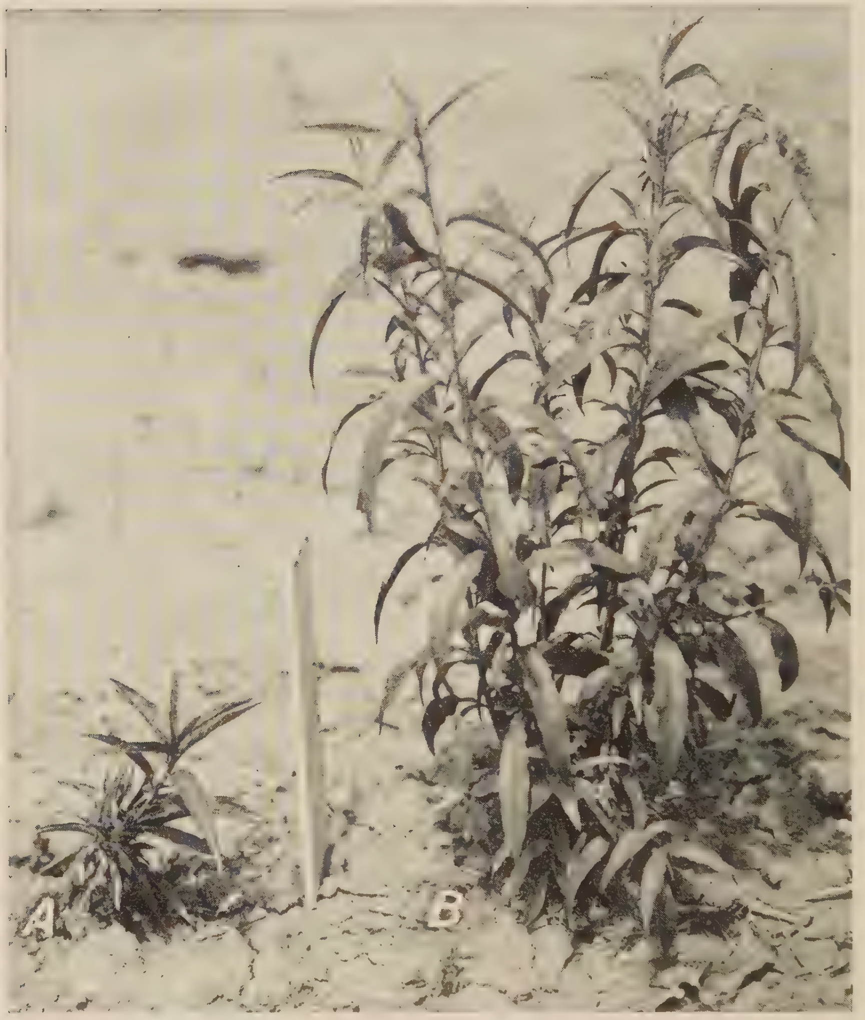

Figure 52.—*A*, **Peach seedling in a nursery row, showing rosetting frequently occurring after inoculation with material affected with sour cherry yellows;** ***B*, comparable uninoculated seedling.**

trees developed chlorotic etch ring leaf symptoms in the year of inoculation. Striking prune dwarf symptoms developed the year after inoculation and in succeeding years on all trees inoculated with buds from Montmorency affected with yellows and known to contain latent necrotic ring spot virus. Prune dwarf symptoms have not been expressed on the trees inoculated with buds carrying the necrotic ring spot virus without yellows or on those inoculated with buds from the tree showing symptoms of yellows and carrying the strain of necrotic ring spot virus that incites symptoms annually on Montmorency. No symptoms of any kind have been expressed on the unbudded control trees.

By budding back to Montmorency from Italian Prune, both necrotic ring spot and yellows were transmitted from trees that had been inoculated originally with buds from yellows-affected cherry; necrotic ring spot only was recovered from those budded originally from the source of necrotic ring spot alone, and nothing was transmitted from the unbudded controls. It is especially noteworthy that the Italian Prune budded originally with Montmorency buds carrying the viruses of yellows and recurrent necrotic ring spot transmitted both yellows and recurrent necrotic ring spot back to Montmorency, even though no prune dwarf symptoms had been expressed on the Italian Prune.

Transmission

Yellows has been transmitted experimentally by budding or other methods of grafting (*121*); in nature it is transmitted by unknown means, probably insects. Cation (*51*) recently reported that this disease can be transmitted through the seeds of mahaleb cherry.

Incubation Period

In the field, symptoms usually appear under favorable climatic conditions within 1 to 2 years after budding or grafting. In the greenhouse, by starting with stocks and scions beginning to break bud and by maintaining suitable temperatures, Moore and Keitt obtained leaf symptoms on Montmorency sour cherry within a minimal period of 6 weeks. Hildebrand (*86*) reported transmission of cherry yellows to peach with symptom expression in 3 weeks.

Thermal Relationships

In the greenhouse leaf symptoms of yellows were not expressed at an approximately constant temperature of 20° C. or higher, but typical ones appeared at 16° or lower. Typical leaf symptoms developed if the night temperature was 16° or lower even though the day temperature was 20° to 28° (*125, 164*). Temperature appears to be a major factor affecting regional and seasonal expression of leaf symptoms of yellows (*125, 159, 165*).

Efforts to inactivate the virus in bud sticks by heat without killing the buds have thus far been unsuccessful. Moore (*161*) transmitted yellows from Montmorency bud sticks 15 cm. long and 2.7 to 3 mm. in diameter that had been immersed in water at 50° C. for 22 minutes.

Control Measures

The first step in control should be the production of yellows-free nursery stocks of superior quality. Since many nurseries supplying sour cherry stocks are located in regions in which high temperature masks cherry yellows leaf symptoms in most years, some indexing procedure is indicated. In Michigan, New York, and Ontario Rasmussen and Cation (*176*), Hildebrand (*84*), and Berkeley (*8*) have indexed sour cherry stocks on peach. In Wisconsin indexing is done on sour cherry incubated in the greenhouse under favorable conditions for yellows expression (*166*).

New plantings should be made at some distance from older orchards and with stocks as free from yellows as possible. Berkeley, Hildebrand, Moore and Keitt, and Rasmussen and Cation recommend that any trees affected with yellows be removed from young plantings and replaced by healthy stock.

General recommendations regarding bearing orchards cannot be made until the research program is further advanced. However, unless information to the contrary becomes available, it is suggested that no roguing and replanting be done in older orchards in which cherry yellows is well established, but that entire blocks be removed when they become unprofitable. It appears generally advisable to rogue yellows-infected trees in bearing orchards in which the number of affected trees is comparatively very small.

Remarks

No case has yet been reported in which a sour cherry tree diseased with yellows was shown beyond doubt to be free from the necrotic ring spot virus. It is possible that sour cherry yellows is incited by a virus complex that includes the necrotic ring spot virus.

There is evidence of the existence of various strains or complexes of yellows and other viruses and of strain differences in sour cherry varieties in their reaction to these virus entities (*9*, *88*, *92*, *177*).

In three cases following transfer of buds from Montmorency sour cherry trees showing typical yellows symptoms to Montmorency, Berkeley and Moore and Keitt obtained symptoms of green ring mottle. The trees from which these buds were taken have not shown green ring mottle.

GREEN RING MOTTLE

By E. J. Rasmussen, G. H. Berkeley, Donald Cation, E. M. Hildebrand,
G. W. Keitt, and J. Duain Moore

Other Common Names of the Disease

The disease has been called green ring yellows also.

Name of the Causal Virus

No Latin binomial has been suggested. The virus may be referred to as the green ring mottle virus.

History and Geographic Distribution

Green ring mottle has been reported in sour cherry orchards in Michigan, New York, and Wisconsin and in Ontario, Canada. It was first observed in Michigan in 1937, in New York in 1938, in Wisconsin in 1942, and in Ontario in 1941. It has been found as a minor disease in several orchards in Michigan, but it has been observed in but one orchard in Ontario. Since the first report in the respective sections there has been but little spread of the disease.

Economic Importance

At present green ring mottle is classed as of minor importance solely because of its rare occurrence.

Host Range

Green ring mottle has been found in nature on sour cherry (*Prunus cerasus*) only. It has been transmitted experimentally to sour cherry variety Montmorency; sweet cherry (*P. avium*) varieties Bing and Napoleon (Royal Ann); and peach (*P. persica*) varieties Elberta, Rochester, Halehaven, and South Haven and seedlings; and mahaleb cherry (*P. mahaleb*) seedlings.

Symptoms

Sour cherry.—Characteristic symptoms appear on leaves of Montmorency cherry 4 to 5 weeks after petal fall as green spots, rings, arcs, or irregular, curved bands ⅛ to ¼ inch in width distributed more or less evenly over a yellow background (pl. 21, *A*). Affected leaves are cast. The period of defoliation lasts for 10 to 14 days, after which affected trees appear to have recovered. Another symptom consisting of scattered chlorotic spots on green leaves may appear. This last-mentioned symptom is especially prominent in the fall, when the green ring phase is absent, but it may appear in the spring in association with the green ring phase. It was observed first by Berkeley in Ontario, but was confirmed by Cation in Michigan.

As a rule the defoliation caused by green ring mottle is not as severe as that associated with sour cherry yellows, though in Ontario Berkeley observed one tree that had as severe defoliation as any seen in that Province in association with yellows. Rasmussen (96) and Cation found that in Michigan defoliation does not recur year after year on the same trees as consistently as it does with yellows. This does not hold apparently in Ontario and Wisconsin, according to Berkeley and Moore and Keitt, since certain trees both in nurseries and in orchards have shown symptoms annually for 5 or 6 years. However, the amount of defoliation varies with individual trees. For instance, five trees in one orchard in Ontario have shown consistent variation in this respect each year. One tree has shown severe defoliation consistently, while the defoliation of the other four has been moderate to mild each year. Nursery trees in the greenhouse in Wisconsin have shown characteristic symptoms for 4 years. Hildebrand stated that defoliation is relatively light in most years and practically absent in certain ones in New York.

Mahaleb cherry.—Pale chlorotic patterns on green mahaleb cherry leaves, similar to the mottling and line patterns sometimes caused by the necrotic ring spot and sour cherry yellows viruses, are induced by the green ring mottle virus. The mottling tends to occur as rather indefinite to definite rings. Affected leaves persist throughout the season. Since the necrotic ring spot virus appears to be associated with the green ring mottle, the symptoms described may have been caused by the former virus.

Sweet cherry.—In Wisconsin, Moore and Keitt found that the Bing variety of sweet cherry is a symptomless carrier. According to Berkeley, inoculated Napoleon trees showed a few leaves with a mild mottle in Ontario. Transfers from these trees back to peach showed that a virus was present in the Napoleon trees. Whether or not this mild mottle was caused by the necrotic ring spot factor was not investigated.

Peach.—When peach seedlings or varieties are inoculated with the green ring mottle virus, they react with mild shock symptoms of delayed foliation, sometimes with and sometimes without varying amounts of twig dieback. They outgrow these symptoms and appear to recover, though in some cases there is a mild stunting of growth. Rosetted shoots such as those associated with sour cherry yellows are lacking. Inoculations with the severe type of green ring mottle virus reported for Ontario resulted in severe symptoms on peach seedlings and in the ultimate death of the Rochester variety.

From the symptom expressions on peach, Moore and Keitt and Berkeley concluded that all the green ring mottle material used in transmission tests in Wisconsin and in Ontario carried a virus of the necrotic ring spot type.

Diagnostic or Unusual Characteristics

The retention of the green coloring in the form of rings or spots on Montmorency cherry leaves and the less drastic effect on peach differentiate green ring mottle from sour cherry yellows. On the other hand, the yellowing and casting of leaves by Montmorency cherry trees at the same period as by trees affected with yellows and the apparent association of a necrotic ring spot virus suggest a close relationship between green ring mottle and sour cherry yellows. The fact that in both Wisconsin and Ontario inoculations with material affected with typical sour cherry yellows gave symptoms of the green ring mottle type supports this contention.

Transmission

Green ring mottle has been induced experimentally by budding only, and its manner of spread in nature is unknown.

Incubation Period

Bud inoculation in the fall produced symptoms the following spring when growth was renewed.

Control Measures

Disease-free nursery stock should be obtained for new plantings. Source Montmorency cherry trees used for bud sticks should be examined in June when symptoms are expressed. The elimination of source trees which transmit symptoms of necrotic ring spot by an indexing procedure should also eliminate green ring mottle. Affected trees in new plantings should be rogued. Control measures in old orchards must await the result of further research.

Remarks

As already stated, green ring mottle has many points of similarity with sour cherry yellows and may be caused by a strain of the yellows virus. A green ring mottle has also been observed by Hildebrand in New York on both Italian Prune (*P. domestica*) and its rootstock, myrobalan plum (*P. cerasifera*). The relationship of these two mottles is under investigation. Hildebrand (*89*) also reported that infection with green ring mottle virus seemed to inhibit infection with the leaf spot fungus, whereas this did not hold for yellows. However, according to Moore and Keitt, there has been no evidence of this phenomenon for green ring mottle in Wisconsin.

PINK FRUIT

By E. L. Reeves and Lee M. Hutchins

Other Common Names of the Disease

Pink cherry.

Name of the Causal Virus

Pink fruit virus.

History and Geographic Distribution

In 1934 the disease was reported in western Washington and symptoms were described (*81*), but transmissibility was not demonstrated. Investigations by Reeves and Hutchins in 1940 (unpublished data referred to by Reeves (*184*)) established that pink fruit is a transmissible virus disease.

Pink fruit is widely distributed in the sour cherry orchards of western Washington from Whatcom County in the north to Clark County in the south. As a result of a brief survey made in 1938, Reeves, Huber, and Baur (*186*) reported 9 percent of the trees to be affected in certain selected orchards in King and Pierce Counties in western Washington. Coe (*66*) reported that an average of 2 percent of the sour cherry trees was found affected with pink fruit in an orchard survey made in six western Washington orchards during 1942. A disorder that appeared somewhat similar was observed in Idaho and reported by Blodgett (*14*).

Economic Importance

Pink fruit, the cause of serious losses in many western Washington sour cherry orchards, is considered second in importance to brown rot, caused by *Monilinia laxa* (Aderh. & Ruhl.) Honey, from the standpoint of reduction in commercial production. There is some seasonal variability in the percentage of fruits that develop severe symptoms of the disease. Generally, 25 percent or more of the fruits are affected on a tree that has exhibited symptoms for 1 year or longer. It is considered impractical at harvesttime to pick the apparently normal fruits and leave the severely affected; thus the result is a total loss of the crop on affected trees.

Host Range

Montmorency is the only variety of sour cherry (*Prunus cerasus*) known to be affected. Possibly other varieties of sour cherries or other species of *Prunus* may be involved, but they have not been adequately tested.

Symptoms

The fruits exhibit the most conspicuous symptoms of pink fruit (pl. 21, *B*), and these symptoms can be most readily observed from about 2 weeks prior to harvest until shortly thereafter. Affected fruits are smaller than normal, insipid to bitter, pinkish yellow to dull pinkish brown, and with internal and external brown necrotic areas in the flesh. The more immature of the affected fruits tend to be conical, and all fruits exhibiting symptoms tend to drop from the tree earlier than those appearing normal.

The leaves of affected trees often have a tendency to fold upward along the midvein with the general result that the trees appear to be suffering from a lack of moisture. Affected trees blossom late in the spring, and growth is suppressed. Late blossoming alone, however, is not an absolute criterion in diagnosing the disease.

Transmission

Transmission has been effected only by grafting or some adaptation of the grafting process and has been demonstrated by using either shoot or root tissues.

Incubation Period

In field tests 15 months was the minimum incubation period, and in many instances the first symptoms appeared 27 months after inoculation.

Control Measures

Records obtained and observations made from 1939 through 1944 indicate that pink fruit has spread very slowly in some western Washington orchards and not at all in others. Since trees do not recover from the disease and the crops from affected trees are of no commercial value, it is considered that a tree-removal program, together with the careful selection of nonaffected propagation wood, should be suggested as a control measure. Because of the slowness with which the disease apparently spreads, it is even possible that a well-directed control program might entirely eradicate the disease.

Fruit symptoms of pink fruit are similar to those of a disease called western X little cherry which occurs on sour cherry in several Western States and which has been related to the western X-disease of peach. Certain differences between pink fruit and this so-called little cherry follow:

1. When Montmorency trees on mahaleb rootstock are inoculated with buds from western-X-little-cherry-affected trees, they wilt and decline; similar trees inoculated with the pink fruit virus exhibit fruit symptoms, but do not wilt.
2. Symptoms of western X-disease developed on leaves of peach trees inoculated with buds from Montmorency trees affected with western X little cherry, but not on those of trees inoculated with the pink fruit virus.
3. The incubation period of pink fruit was found to be at least 15 months and often it was 27 months, but the incubation period of western X little cherry on sour cherry is 9 months, when inoculations are made in the fall.

Further work needs to be done in comparing pink fruit and western X little cherry to ascertain whether a relationship exists between these two diseases.

NECROTIC RING SPOT

By G. H. Berkeley, Donald Cation, E. M. Hildebrand,
G. W. Keitt, and J. Duain Moore[41]

Other Common Names of the Disease

Ring spot, shredded leaf, necrotic leaf spot, and small leaf.

Name of the Causal Virus

Necrotic ring spot virus.

History and Geographic Distribution

In Ontario, Canada, necrotic ring spot was first observed in 1939 and its virus nature was demonstrated in 1940 when typical symptoms were expressed on Montmorency sour cherry and a variety of duke cherry as a result of inoculations by budding from infected trees (*250*). At about the same time sour cherry necrotic ring spot, or a closely related disease, was discovered independently in New York, Wisconsin, Pennsylvania, and Michigan.

Economic Importance

The data on economic importance are not conclusive. In Wisconsin, according to Moore and Keitt's report, the common strains of necrotic ring spot virus have not been shown to have any harmful effect on the fruiting habit or yield of Montmorency sour cherry except possibly in the year when acute symptoms are severe (*252*). In Ontario, there are indications that necrotic ring spot apparently has some reducing effect on yield, and the thinness of the foliage of affected trees would appear to support these indications. From records on yields of healthy trees and of those affected with necrotic ring spot in three orchards, Berkeley noted a gradual reduction in one orchard but no appreciable consistent reduction in the other two. In New York increased susceptibility to winter injury was associated with the presence of necrotic ring spot (*90*). In Michigan, where the disease is prevalent in most orchards, no reduction in leaf size or indications of reduced yields have been apparent, but comparisons of affected with virus-free stock have not been made.

Host Range

The disease has been found naturally on sour cherry (*Prunus cerasus*) and chokecherry (*P. virginiana*). It has been transmitted by budding or grafting to sweet cherry (*P. avium*) seedlings and varieties Bing, Napoleon (Royal Ann), Black Tartarian, Giant, and Eagle; to mazzard (*P. avium*) seedlings; to peach (*P. persica*) seedlings and varieties Elberta, Rochester, and Halehaven; to domestica plum (*P. domestica*) varieties Bradshaw, Italian Prune, and Lombard; to American plum (*P. americana*) seedlings; to various wild and ornamental species of *Prunus:* Bessey cherry (*P. besseyi*), a Hansen bush-cherry, sand cherry (*P. pumila*), pin cherry (*P. pensylvanica*), and chokecherry (*P. virginiana*); to duke cherry (*P. avium* × *P. cerasus*) varieties Royal Duke and Grand Duke; to myrobalan plum (*P. cerasifera*) and mahaleb cherry (*P. mahaleb*) rootstocks; and to sour cherry (*P. cerasus*) varieties Montmorency, Early Richmond, and English Morello. Cucumber (*Cucumis sativus* L.) was infected from sour cherry by rubbing juice on it.

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[41] Senior authorship of this section is by mutual agreement and not by priority of publication.

In the Wisconsin host-range studies (*163*) the following hosts in the list just given were not tested by Moore and Keitt: *P. avium* varieties Giant and Eagle; *P. persica* seedlings; and the Grand Duke variety of *P. avium* × *P. cerasus*. Necrotic ring spot was transmitted by budding back to Montmorency, with the expression of typical symptoms, from all other hosts listed except *P. pumila* with which no transmission was attempted.

All attempts by Moore and Keitt to transmit necrotic ring spot from Montmorency sour cherry to the Japanese plum (*P. salicina*) varieties Abundance and Burbank failed. No symptoms were produced on these two varieties, and necrotic ring spot was not recovered from them by budding back to Montmorency.

After the first year of symptom expression, most of the hosts studied by Moore and Keitt carried the virus without further symptom expression. No host of necrotic ring spot virus that did not at some time express symptoms has yet been found, and most hosts carrying necrotic ring spot virus and not expressing symptoms failed to develop symptoms when inoculated with necrotic ring spot virus.

Symptoms

Sour cherry.—The initial effect of necrotic ring spot on the Montmorency variety is a pronounced delayed foliation of individual limbs or entire trees (fig. 53, *A*, *B*). Leaves on affected branches are reduced in size, and before they unfold they may show light-green spots and dark rings, which vary in size from 1 mm. or less up to 5 mm. in diameter and have a water-soaked appearance (fig. 54, *B*, *C*). Partial rings and sometimes concentric rings are also present. The surface of affected leaves is roughened, and the margins tend to be wavy (*9*). In the early stages the ring symptoms are visible by reflected light only. As the season advances, the affected areas may become necrotic, fall out, and give a "shredded leaf" effect (fig. 54, *A*). Symptom expression usually is limited to the first leaves that unfold; leaves formed later generally do not show symptoms. In Ontario Berkeley observed that affected trees have a reduced number of leaves and therefore have a thin appearance.

Sometimes blossom symptoms also may be present on severely affected branches. The pedicels may be shortened until they are almost sessile, according to Moore and Keitt and Berkeley. In severe cases, the calyx and corolla may be twisted and distorted and sometimes chlorotic or necrotic rings or arcs are present in the sepals. Moore and Keitt noticed also that such severely affected blossoms ordinarily do not set fruit. Occasionally fruits with small rings similar to those on the leaves are found on trees showing severe necrotic ring spot symptoms on the leaves.

The general experience has been that trees severely affected one year show few or no symptoms in subsequent years except in Ontario where the thinness of foliage has been characteristic. Usually, if severe symptoms are present only on certain branches the first year, other branches may show striking symptoms the next year. In Wisconsin no branches of orchard trees have been observed to show striking symptoms in more than one year, but certain nursery trees have shown striking ring symptoms and wavy leaf margins but no necrosis each year since the initial symptoms appeared in 1941. In a few trees, however, ring symptoms have persisted for 4 years in Ontario and for 6 years in New York. This variation in symptom expression is indicative of strains of the virus, or possibly of strains of the host (Montmorency cherry), or of both.

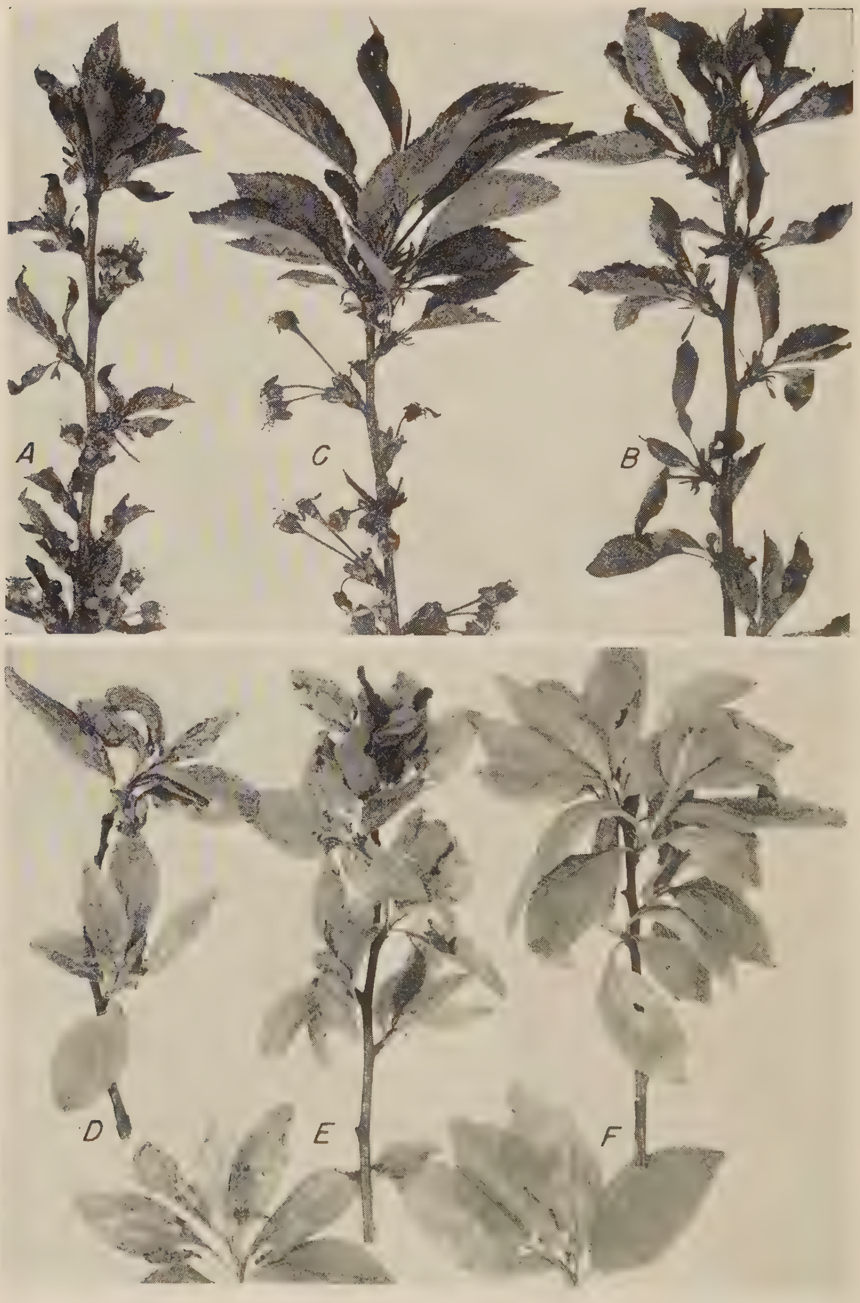

Figure 53.—*A* and *B*, Branches of Montmorency cherry affected with necrotic ring spot, showing delayed foliation, stunted leaves, and shortened blossom pedicels; *C*, comparable healthy branch of Montmorency cherry; *D* and *E*, Italian Prune branches affected with necrotic ring spot, showing necrotic spotting of leaves and stunting; *F*, comparable healthy branch of Italian Prune variety.

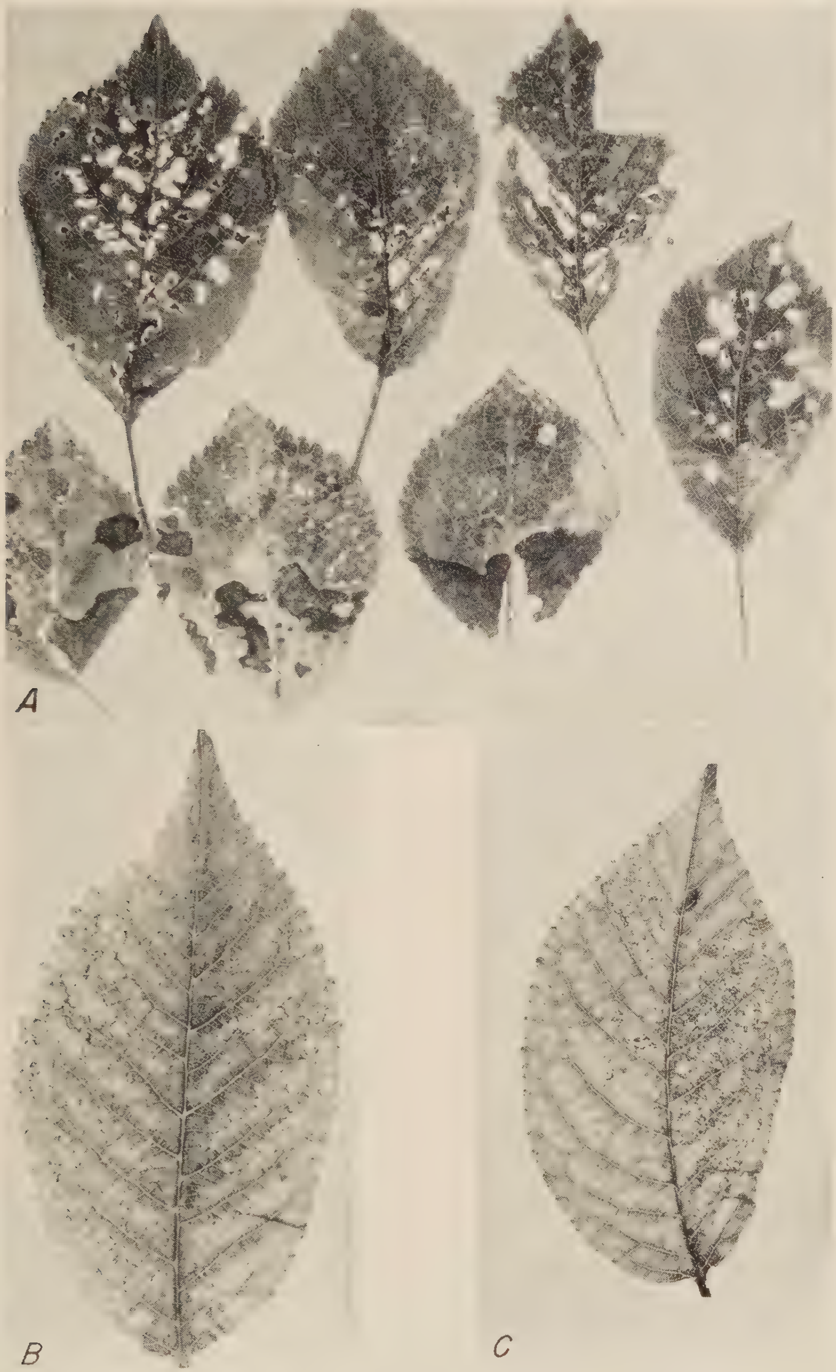

Figure 54.—Montmorency cherry leaves affected with necrotic ring spot, showing *(A)* severe necrotic shock symptoms and *(B* and *C)* water-soaked pattern consisting of rings and arcs of rings.

Under greenhouse conditions very severe and striking typical symptoms were obtained by Moore and Keitt on English Morello sour cherry, beginning about 10 days after inoculation. The early water-soaked type of rings, necrotic spotting, and general necrosis, followed by severe leaf shredding, were present.

Sweet cherry.—In field inoculations of Bing and Black Tartarian sweet cherry chlorotic rings and chlorotic spotting were obtained on only a part of the foliage. Some leaf shredding sometimes occurred. In Ontario there was a tendency for the margins of affected leaves to be wavy. When inoculations were made in the greenhouse, symptoms were obtained by Moore and Keitt in 6 to 8 weeks; these symptoms were expressed as chlorotic ring spotting or chlorotic and necrotic spotting followed by shredding.

In New York symptoms similar to those on Black Tartarian were obtained on the Eagle variety by Hildebrand. Moore and Keitt reported that under greenhouse conditions symptoms on Napoleon were about the same as those on Bing except that there was in addition some killing of spurs. In field inoculations in Ontario no definite symptoms were obtained on Napoleon even though transfers back to peach indicated that the necrotic ring spot virus was present in the inoculated trees. Symptoms obtained by Moore and Keitt and by Hildebrand on mazzard seedling were about the same as those on Bing.

Mahaleb cherry.—Chlorotic rings, spots, and patterns were obtained by Berkeley, Hildebrand, and Moore and Keitt.

Duke cherry.—Very severe and striking typical necrotic ring spot symptoms similar to those on English Morello occurred on Royal Duke cherry. Most budded trees of this variety, however, expressed both the ring symptoms and the necrotic spotting for 2 years after budding with material affected with certain forms of necrotic ring spot, according to Moore and Keitt and Berkeley.

Peach.—Elberta and Rochester trees inoculated in the fall generally had dead branches above the bud insertion by spring. Often entire trees, especially those of the Rochester variety, were killed. When branches were not dead above the point of bud insertion before start of growth in the spring, there was generally a severe delayed foliation followed by death. Dieback was common on other branches, and bark splitting and bark cankers often were present on the trunk and larger branches of inoculated trees. Parts of trees that survived appeared to be fairly normal by the end of the growing season, though some mottling of foliage sometimes occurred. According to Berkeley (*8*), Hildebrand, and Cation, similar symptoms occurred on peach seedlings.

There was considerable variation as to degree of dieback and killing of peach resulting from inoculation with necrotic ring spot virus from different sources. For instance, Berkeley, Hildebrand, and Cation found that virus from some sources killed entire trees while that from others produced mild dieback. Hildebrand found that virus from still others produced no killing or dieback whatever on peach, though it might cause mottling of the leaves (*91*). At present this variation in response is considered to be due to strains of the necrotic ring spot virus or possibly to contamination with other viruses. By making inoculations under greenhouse conditions on Elberta just at bud break, Moore and Keitt found that chlorotic and necrotic spotting occurred shortly after the leaves unfolded and that most affected leaves were abscised within a period of several weeks. In some cases entire tops of trees were killed above the point of bud insertion. After the period of initial shock new leaves were for the most part free of symptoms. On the Rochester variety there was only a faint, chlorotic spotting, but no necrosis or leaf casting. There was, however, more shoot killing than on Elberta or Halehaven, and this killing was limited almost entirely to terminals.

Plum.—Symptom expression was obtained by Moore and Keitt about 8 weeks after they had budded American plum and the Bradshaw variety of domestic plum in the greenhouse; small, chlorotic or necrotic flecks which tended to follow the larger veins appeared. According to Berkeley, numerous, small, necrotic spots or flecks appeared on unfolding leaves the year following inoculation of Italian Prune (fig. 53, *D, E*). Affected branches showed slight delay in foliation, and affected trees outgrew the symptoms and later appeared to be normal.

Pin cherry.—In a greenhouse with a constant temperature of approximately 21° C. at Wisconsin and under greenhouse conditions in Ontario, symptoms on pin cherry consisted of severe necrosis and shredding of leaf tissue. Recovery after initial shock was striking in this species.

Ornamentals.—In the stone-fruit-virus greenhouse at Wisconsin (temperature range at night 13° to 15° C.) no symptoms were obtained on *P. besseyi,* and only mild symptoms occurred on *P. pumila* and *P. virginiana.* In greenhouses with approximately constant day and night temperatures of 24° or 28° striking chlorotic ring spots or line patterns occurred on all three species. In addition, *P. besseyi* showed chlorotic spotting, leaf roughening, and some necrotic spotting, and *P. pumila* showed necrotic spotting, and shredding (*164*).

Transmission

The disease can be transmitted by any grafting method. Symptoms generally show early in the spring following fall budding. In one case symptoms were observed by Berkeley in September on the new leaf growth of the Italian Prune variety bud-inoculated in August. The Wisconsin investigators (*162*) successfully transmitted necrotic ring spot by mechanical means from sour cherry to cucumber.

Incubation Period

In greenhouse work with stocks and scions just about to break dormancy, symptoms were obtained by Moore and Keitt in from 5 days on Montmorency sour cherry to 8 weeks on Bradshaw and American plum. In Hildebrand's tests on sour cherry the incubation period ranged from 10 days to 5 weeks and on sweet cherry from 2 to 6 weeks. On Italian Prune and peach seedlings the incubation period was 2½ to 5 weeks, according to Berkeley.

Thermal Relationships

Necrotic ring spot has been transmitted, with the expression of symptoms, from Montmorency to Montmorency sour cherry over a temperature range of 10° to 28° C., with much more necrosis and shredding of leaves at the higher temperatures. The best temperature for expression of both rings and necrosis on Montmorency was 20° to 24°. Killing of entire terminals and spurs was common at 28°. Studies on inactivation of the virus in Montmorency bud sticks indicate that a heat treatment sufficient to inactivate the virus will kill the cherry tissue (*161*). Necrotic ring spot was transmitted by Moore and Keitt from bud sticks 6 inches long and 2.7 to 3.0 mm. in diameter that had received a heat treatment of 50° for 22 minutes.

Control Measures

Cherry trees to be used as sources of budwood should first be indexed on peach or virus-free Montmorency sour cherry in order to ascertain whether the necrotic ring spot virus is present. Buds from healthy trees only should be used for top-working or for general propagation purposes.

In some States and in Ontario, Montmorency sour cherry nursery stock free from the necrotic ring spot virus and the cherry yellows virus is now, or will soon be, available. Such nursery stock is recommended for planting purposes.

Remarks

The fact that Montmorency sour cherry is often a symptomless carrier of the necrotic ring spot virus makes the problem of evaluating the economic importance of necrotic ring spot very difficult. Moreover, the possible effects of the necrotic ring spot virus on symptom expression of sour cherry yellows and on yield from trees affected with yellows are not known at present, since a virus of the necrotic ring spot type or a necrotic ring spot factor has been associated with yellows from all sources investigated to date. No one has yet been able to separate the necrotic ring spot factor from the yellows complex.

The problem of their detection in nursery stock has been approached in two ways. In Wisconsin (*166*) reliance has been placed chiefly on the reaction of disease-free Montmorency sour cherry to inoculation, whereas in New York (*95*), Michigan, and Ontario peach seedlings have been used as indicators. The latter procedure has been used on the grounds that the peach when not cut back reacts to necrotic ring spot virus with delayed foliation, varying degrees of dieback, and necrotic spotting of leaves and to sour cherry yellows virus with varying degrees of rosetting occasioned by shortened internodes in addition to the afore-mentioned symptoms associated with necrotic ring spot.

VIRUS DISEASES OF PLUM AND PRUNE

PRUNE DWARF

By E. M. Hildebrand, T. B. Lott, and R. S. Willison

Other Common Names of the Disease

Prune mosaic, Fellenberg mosaic, willows, and shoestring of Italian Prune.

Names of the Causal Virus

Nanus pruni Holmes; Prunus virus 6 (Thomas and Hildebrand) Smith.

Geographic Distribution

Prune dwarf is known only in New York and in Ontario and British Columbia, Canada.

Economic Importance

Prune dwarf renders Italian Prune (Fellenberg) unproductive, but it is of minor importance because of its limited distribution. Since two strains of the virus occur on sour and sweet cherries in New York (*85, 92*) and others on sweet cherry in Ontario (*7, 246*), the economic importance may prove greater than is now realized.

Host Range

Characteristic strap-shaped leaves are produced on Italian Prune and Lombard plum (*Prunus domestica*); and various symptoms are produced on peach (*P. persica*) seedlings and varieties and on mahaleb cherry (*P. mahaleb*) rootstock. The virus may occur without causing symptoms in damson plum (*P. insititia*); in domestica plum (*P. domestica*) varieties Bradshaw, Reine Claude, and German Prune; in Japanese plum (*P. salicina*) variety Abundance; in Shiro hybrid plum; in myrobalan plum (*P. cerasifera*) rootstock; in sweet cherry (*P. avium*) varieties Napoleon (Royal Ann), Black Tartarian, Eagle, and Elkhorn; in sour cherry (*P. cerasus*) variety Montmorency; and in apricot (*P. armeniaca*) variety Niagara. The disease has been found in nature on Bradshaw and damson plums, Italian Prune, and sweet and sour cherries. The remainder of the hosts have been experimentally infected.

Symptoms

Plum.—The affected leaves of the Italian Prune variety (fig. 55, *A*) are small, narrow, straplike, rugose, somewhat thickened, distorted, glazed, frequently emarginate, and sometimes marbled with an obscure mosaic mottle (*232*). The onset of the disease on Italian Prune is marked by a relatively mild chlorosis (rings and mottling) on normal-sized leaves. Leaves formed later on the same tree are smaller and spindly. Terminal growth is reduced, and internodes are shortened. Unless the trees are small and young, they do not usually show symptoms over the entire tree. In the first year after inoculation the diseased shoots may be scattered among branches that appear normal. Later occasional healthy shoots may occur on trees completely and severely affected. Diseased trees blossom well, but the pistils are aborted and few fruits mature. The petals of affected flowers are frequently narrow and distorted (*85, 232*). Fruits on diseased trees are often larger than normal and of excellent quality. Willison (*246*) obtained several strains of the virus from damson plum, Bradshaw plum, sweet cherry, and sour cherry. These sources vary in their virulence and effect on Italian Prune and in their rate of spread through the tree.

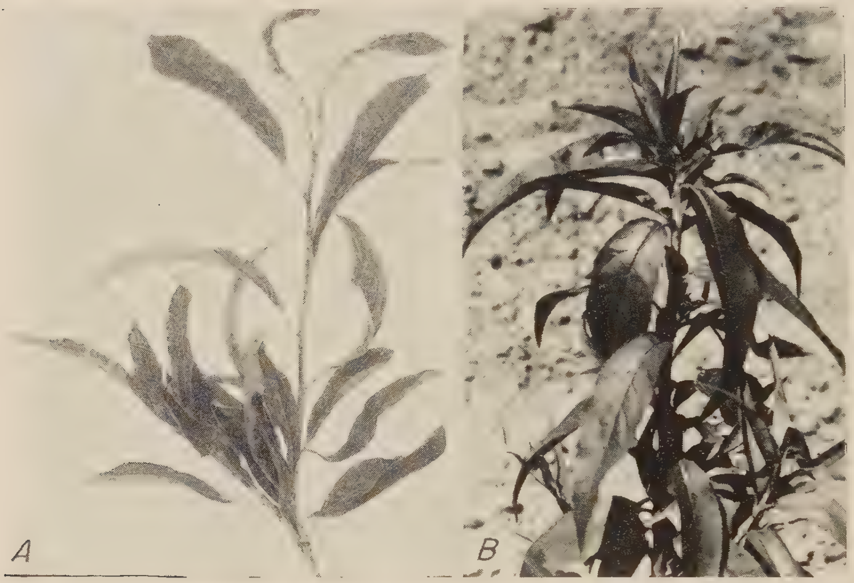

Figure* 55.**—A*, Italian Prune branch affected with prune dwarf, showing strap-shaped leaves; *B*, peach shoot affected with the same disease, showing short internodes and spikelike apical leaves.**

Foliage symptoms on Lombard plum are similar to, but less severe than, those described for Italian Prune. There may be mild, chlorotic flecking of early leaves, waviness of the laminae, and dwarfing and semirosetting of the shoots (*85, 246*). Reduction in yield is slight (*85*). With the milder virus strains there seems to be a tendency toward localization of the disease in the vicinity of the point of inoculation (*246*).

Other plum varieties may show transient symptoms such as mild mottle, chlorotic spotting, ring patterns, and delayed foliation in the early stages of infection; apparently they recover later, though in some instances a slight dwarfing effect may persist.

Peach.—Only minor differences were noted in the symptom expressions of individual virus strains on peach varieties. However, some strains caused more severe symptoms than others. Prune dwarf on peach is characterized by a slight delay in foliation, wavy leaf margins, and a tendency for the leaves to roll upward and inward from their margins and to be stiff and erect, pointing upward. Dwarfing of shoots and shortening of internodes (fig. 55, *B*) sometimes resulted in loose rosettes, but the degree of dwarfing and shortening varied with the different strains of the virus. Leaves produced early in the season were sometimes marked by transient mottles, chlorotic spotting, feathery mottled patterns, or confluent rings. Superficial bark necrosis was noted with some strains. Milder strains of the virus produced slight symptoms that usually became masked after the initial stage. Lott observed that symptoms persist on peach for 2 or 3 years. Some peach trees may show very little effect.

Cherry.—Symptoms on sweet cherry varieties consisted mainly of rings of varying size and intensity, of line or oak-leaf patterns, and of mottling on early-spring leaves. Ordinarily the symptoms were largely masked after the first season of infection. Field data on yield indicated that the crop is definitely reduced by prune dwarf.

In early stages of infection of the Montmorency variety of sour cherry there was a tendency for delayed foliation (*246*). Also some early leaves showed various degrees of ring spotting, necrosis, and flecking. These symptoms were most prominent on Montmorency inoculated with a mixture of prune dwarf and line pattern viruses. Late in the season the symptoms sometimes entirely disappeared. They seldom recurred subsequently. In New York one strain of prune dwarf virus from a yellows-affected cherry tree sometimes induced symptoms simulating yellows when passed through Italian Prune and returned to Montmorency cherry. In Ontario a strain of pure prune dwarf virus contaminated with the line pattern virus caused coarse line pattern symptoms later associated with necrotic spots and streaks on a considerable number of leaves scattered through a Montmorency cherry tree. The symptoms recurred in mid-June; affected leaves were usually cast.

The reaction of mahaleb cherry seedlings to each strain was variable. Some seedlings were symptomless and others exhibited mottles, lines, coarse rings, and oak-leaf patterns on leaves produced in spring and late summer (*246*).

Apricot.—On apricot no leaf symptoms beyond faint mottling were observed, but foliation was sometimes delayed in the spring and some reduction in vigor sometimes occurred (*246*).

Diagnostic or Unusual Characteristics

The dwarfed, irregular, strap-shaped leaves of Italian Prune and Lombard plums and the wavy, stiff leaves and shortened internodes of peach are important diagnostic characters. The occurrence of occasional normal twig growth on diseased branches and the slow movement of the virus in plums are also significant. In Ontario the disease occurs on Italian Prune variety only when that variety has been top-worked on damson plum, indicating that the damson is a carrier. According to Hildebrand, thermal inactivation of the prune dwarf virus has an end point at 50° C. in 20 to 22 minutes.

Transmission

The various strains of prune dwarf virus are readily transmitted by budding and grafting. The insect vector is not known though limited natural spread has been observed in New York (*85*). Lott transmitted a strain of the virus from Napoleon sweet cherry which produced very slight symptoms on Italian Prune soon after grafting when transmission was made direct from cherry to Italian Prune or through peach.

Incubation Period

When inoculation occurs early in the growing season, the incubation period may be as short as 5 weeks on Italian Prune. When trees are inoculated in the fall, the symptoms usually do not appear until the following spring. Symptom development may be delayed until the second growing season. When seedling peaches are inoculated by budding and cut back to the infective bud, symptoms of chlorosis may develop within 1 month on the first new growth of the seedling.

Control Measures

Roguing of affected Italian Prune is recommended as such trees are commercially unprofitable. When Italian Prune is to be on other, top-worked plum varieties, it is desirable that these varieties first be indexed to determine whether the virus is present and that infected ones be discarded. Indexing can be done by placing buds from the trees in question on healthy susceptible nursery trees or by placing healthy Italian Prune scions on the trees to be top-worked.

Remarks

In New York inoculum containing strain 1 of sour cherry yellows virus induced symptoms of prune dwarf on Italian Prune and Lombard plum varieties and also a rosette on peach, as did a mixture of cherry yellows and necrotic ring spot viruses in Ontario. Willison has observed no definite instances of yellows symptoms on Montmorency sour cherry inoculated with prune dwarf virus from naturally affected Italian Prune. The symptoms of prune dwarf on peach resemble those of rosette mosaic, but the relationship of the respective causal viruses has not yet been demonstrated. The prune dwarf virus has been found in sweet cherry associated with the tatter leaf virus and in Italian Prune associated with the line pattern virus. Though the reaction of peach to the prune dwarf virus strains worked with in Ontario indicated that these strains were not contaminated with necrotic ring spot virus, there is no reason why this combination of viruses should not occur in nature. Indexing trials indicate that prune dwarf may be distributed in masked form in sweet cherry. It is of interest that the thermal-inactivation point of the rosette mosaic virus is near if not identical with that of prune dwarf virus: namely, an exposure of 20 to 22 minutes at 50° C.

PRUNE DIAMOND CANKER

By Ralph E. Smith and H. Earl Thomas

Name of the Causal Virus

Prune diamond canker virus.

History and Geographic Distribution

The disease was noted only occasionally before 1915. Trees planted in the decade after that year often developed a rather high eventual incidence of disease, particularly in the Santa Clara and Napa Valleys of California. It seems now that the prevalence of the disease in the prune-growing districts of the State is related chiefly or entirely to the accidental dispersal of the virus in nursery stock (*4*). At any rate the disease is rare in the Sacramento and San Joaquin Valleys and probably is unrecognized outside the State.

Economic Importance

Affected trees undergo an initial seemingly stimulating effect. This is followed by a slow decline in vigor and fruitfulness hastened by secondary causes. Eventually diseased trees become worthless or nearly so. The widespread occurrence of the disease expressed and in the incubation stage has been the cause of substantial losses in French (Agen) prune orchards and will continue to be for some years to come.

Host Range and Varietal Susceptibility

Symptoms of diamond canker are expressed only on the French (Agen) prune (*Prunus domestica*). The disease appears to be more prevalent in certain horticultural selections of French prune than in average trees of the variety. Some of these selections have become widely planted. Typical cankers developed on the tops grown from diseased French prune scions grafted on apricot (*P. armeniaca*), almond (*P. amygdalus*), peach (*P. persica*), myrobalan plum (*P. cerasifera*), and other rootstocks but without symptoms on the rootstocks. In a small percentage of cases the virus passed from diseased scions of French prune grafted on one side of a non-symptom-producing stock to previously healthy arms of a French prune grafted on the other side (*214*).

Symptoms

The name "diamond canker" arose from the fact that in some cases the bark becomes thickened and roughened in a more or less diamond-shaped area often around a wound (*212*). The wood may or may not be exposed. In other cases the bark may be roughened generally over the older parts of trees (fig. 56). Slight roughening is sometimes seen on bark only a year or so old. It is not known whether the variations are due to strains of the virus, to different susceptibility of the prune clones, or to both. No fruit or leaf symptoms have been recognized.

Transmission and Incubation Period

Healthy scions grafted on affected trees may show symptoms in 2 years, but often do not do so until several years later (*214*). For this and other reasons there is some uncertainty whether there is natural spread in orchards. The evidence suggests that most if not all dissemination has been by buds and scions from shoots too young to show recognizable symptoms.

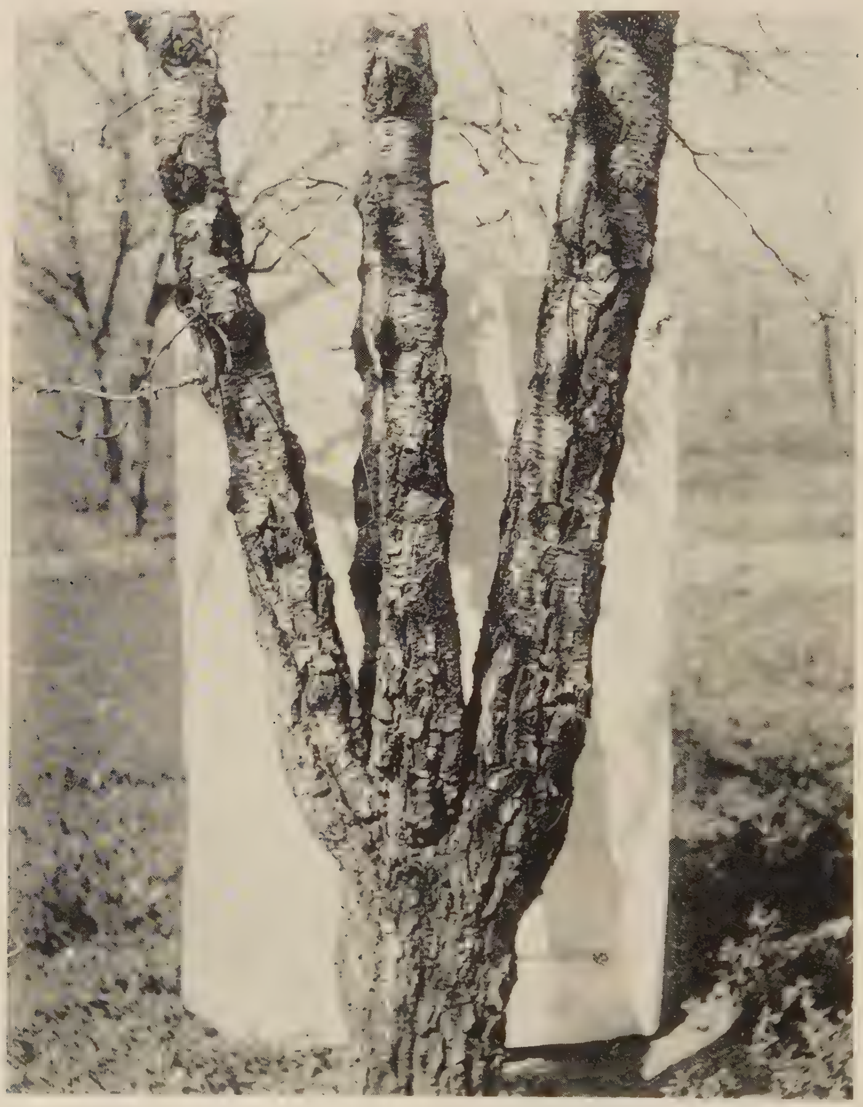

Figure 56.—French (Agen) prune affected with diamond canker.

Control Measures

Since there is little evidence of spread of the virus causing diamond canker from tree to tree in the orchard, the disease should be eliminated by removal of diseased trees as they become unproductive and replacing them with nursery stock propagated from trees known to be free from the virus.

Remarks

It is suggested that prune diamond canker is similar to psorosis of citrus (*72*), but it is not known whether the causal virus of diamond canker like that of psorosis is systemic and gives rise to a localized form that causes localized cankers. In any event the diamond canker virus can be recovered from symptomless parts of an affected tree.

LINE PATTERN

By Donald Cation, G. H. Berkeley, J. A. Milbrath,
R. S. Willison, and S. M. Zeller

Other Common Names of the Disease

Shiro line pattern, peach line pattern, and banded chlorosis of oriental flowering cherry.

Names of the Causal Virus

Marmor lineopictum was applied by Cation to line pattern of plum and *M. pallidolimbatus* was applied by Zeller and Milbrath to banded chlorosis of oriental flowering cherry. Ultimately Zeller and Milbrath considered banded chlorosis closely related to or identical with line pattern. Therefore, by priority, *M. lineopictum* Cation is now the recognized name.

History and Geographic Distribution

Line pattern has been reported on plum in the Niagara district of Ontario, Canada, (*7, 247*) and in Michigan (*47*), New York (*85*), and Kentucky (*242*) and as banded chlorosis on oriental flowering cherry in Washington and Oregon (*261*). The disease has been identified on plum nursery stock grown in Ohio (*47*), Kentucky (*242*), Oregon, and Washington (*261*). Similar diseases have been reported in California (*233*) and Europe (*2, 3, 53*). Line pattern is probably widely distributed at least throughout the United States. Symptoms of line pattern on plum and peach were first reported by Valleau (*242*). He did not assign a name to the disease, and there is the possibility that in some instances he was working with a mixture of viruses (*47*).

Economic Importance

Line pattern on most varieties of plum and peach appears to be of negligible importance, and on oriental flowering cherry damage is limited to unsightly foliage. There are indications, however, that the severity of symptoms increases when the line pattern virus is combined with other viruses and a complex infection results.

Host Range and Varietal Susceptibility

Line pattern occurs in nature frequently on Japanese plum (*Prunus salicina*) varieties Red June and Abundance and on oriental flowering cherries (*P. serrulata*) varieties Amanogawa, Shirotae (Mount Fuji), Naden,[42] Kwanzan, and Temari and to a less extent on peach (*P. persica*) seedlings.

The following species and varieties have been artificially infected: Peach varieties Elberta, J. H. Hale, Rochester, and Carman and seedlings; Japanese plum varieties Abundance and Red June; the hybrid plums Shiro and First; domestica plum (*P. domestica*) varieties Italian Prune, Early Golden Drop, German Prune, Imperial Gage, Reine Claude, Lombard, and Grand Duke; myrobalan plum (*P. cerasifera*) seedlings; sweet cherry (*P. avium*) varieties Black Tartarian and Napoleon (Royal Ann); mazzard (*P. avium*) seedlings; sour cherry (*P. cerasus*) variety Montmorency; and mahaleb cherry (*P. maha-*

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[42] The varietal name "Naden," as used in this paper, refers to an apparent horticultural strain of the variety Kwanzan. It should be noted also that the name "Naden" is sometimes applied to the variety Takasago (*P. sieboldii*).

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leb) seedlings. On these artificially infected hosts the symptoms varied in intensity. Apricot (*P. armeniaca*) variety Niagara appeared to be a symptomless host.

Symptoms

The symptoms are almost invariably confined to the leaves emerging in the spring when the daily mean temperatures are below 55° to 60° F. (12.8° to 15.6° C.), and they persist on these leaves throughout the season. Therefore, later in the summer most of the leaves on infected trees are symptomless.

Plum.—Commercial varieties of plum were very variable in symptom expression. Patterns on leaves usually consisted of lines, bands, and oak-leaf patterns. Vein banding was observed by Willison and Berkeley. In 1945 Zeller and Milbrath noted small rings in addition to other symptoms.

On Shiro plum (fig. 57, *A*) and myrobalan plum seedlings (fig. 58, *D*) the patterns were conspicuously yellow or yellow green in the spring and became a vivid creamy white early in the summer (*247*).

On Abundance plum the patterns resembled those on Shiro, but were less conspicuous and more transient (*247*). The symptoms consisted of chlorotic rings, spots, and line patterns bordered with a white to bright-golden band, which was more pronounced for line pattern than for banded chlorosis, according to Zeller and Milbrath. Infected Abundance bore normal crops of good-quality fruit (*47*).

On Italian Prune, Reine Claude, and First plums the patterns were of a very faint oak-leaf type; on Italian Prune they were absent in some seasons. On German Prune and Grand Duke plums fine, irregular, yellowish lines predominated (*247*). Willison and Berkeley found that Imperial Gage and Early Golden Drop plums and some myrobalan plum seedlings carried the virus without symptoms. Other myrobalan plum seedlings showed a golden-net pattern bordering the veins and veinlets, according to Zeller and Milbrath. On Lombard plum only a few leaves were affected. On these, Willison and Berkeley noted faint markings of the Shiro type, but the more characteristic symptom was a regular, chlorotic, and eventually necrotic, line outlining the marginal triangular section on the lamina and usually distorting the leaf.

Peach.—No differential reaction of peach varieties to line pattern was detected by Willison. The characteristic symptom was a fine, irregular, wavy band on each half of the lamina, usually forming a symmetrical pattern (fig. 57, *B*). Some leaves were marked with a network of fine lines, or a golden-net pattern, fine confluent rings, vein banding, or an oak-leaf pattern, or both of the last two. Markings on peach leaves were generally pale green or more rarely yellow green, and usually they disappeared in the course of the summer. The number of affected leaves and the predominant type of symptom varied in outline from year to year in response to the temperature conditions in the spring (*247*). Similar line patterns and ring spots were produced on J. H. Hale peach inoculated by Milbrath and Zeller with buds from trees affected with line pattern or with banded chlorosis.

Sour cherry.—On Montmorency sour cherry the symptoms in the early stages of infection were inconspicuous, pale-green bands, spots, and coarse rings, and a few necrotic spots also were present. In the following year sharply defined lines (fig. 58, *B*) did not appear until early June. These lines were sometimes chlorotic, but more commonly they appeared as watermarks seen best by transmitted light, according to Willison and Berkeley.

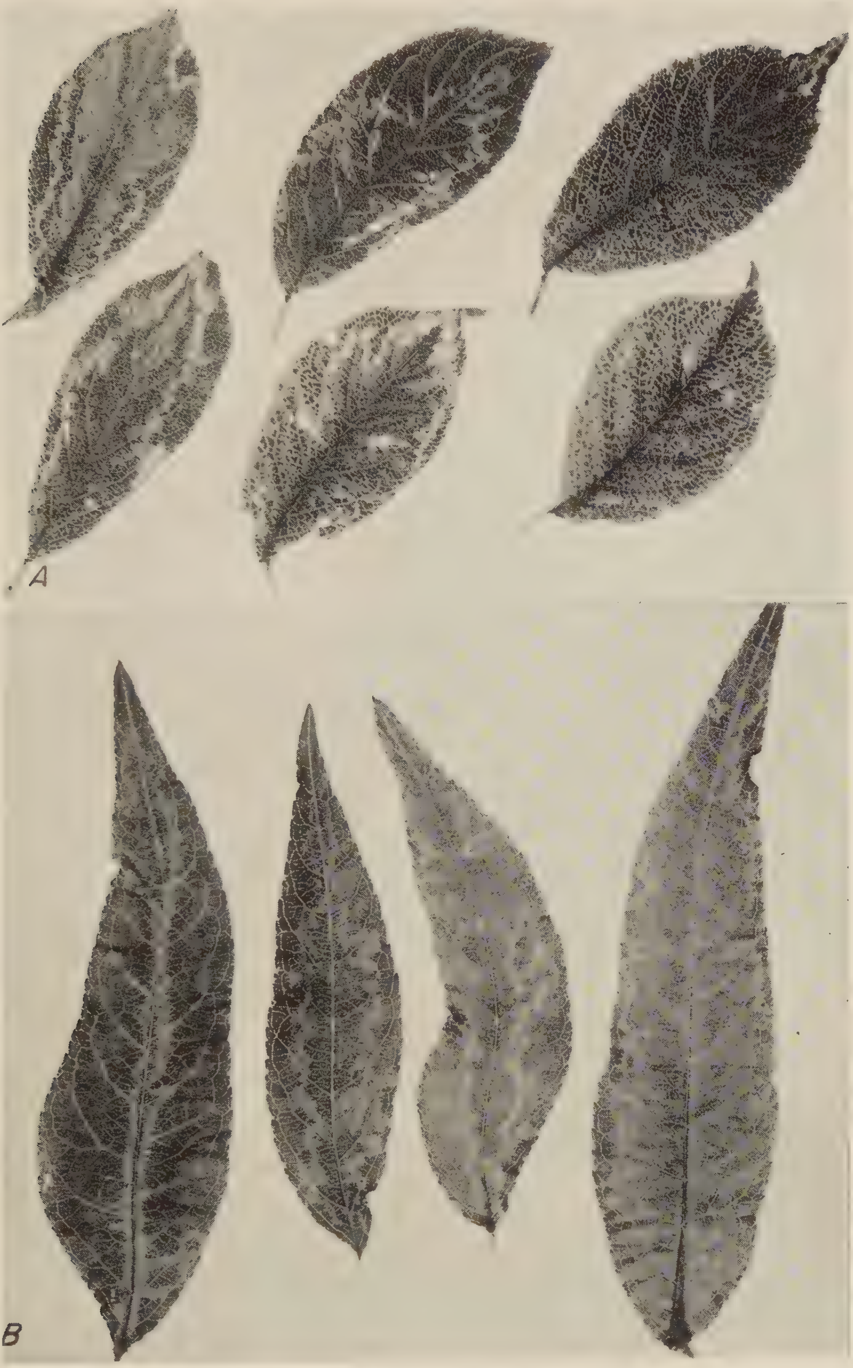

Figure 57.—*A*, Shiro plum leaves affected with line pattern; *B*, peach leaves affected with the same disease.

Mahaleb cherry.—On mahaleb cherry seedlings symptoms consisted of oakleaf patterns and coarse lines and rings, sometimes darker and sometimes paler than the rest of the leaf (fig. 58, *C*). Short, narrow yellow bands along the larger veins are of diagnostic value. In these areas the veins, and frequently part of the yellow-band tissue, eventually become necrotic and either split or drop out. The severity of symptoms varied with the individual trees (*247*).

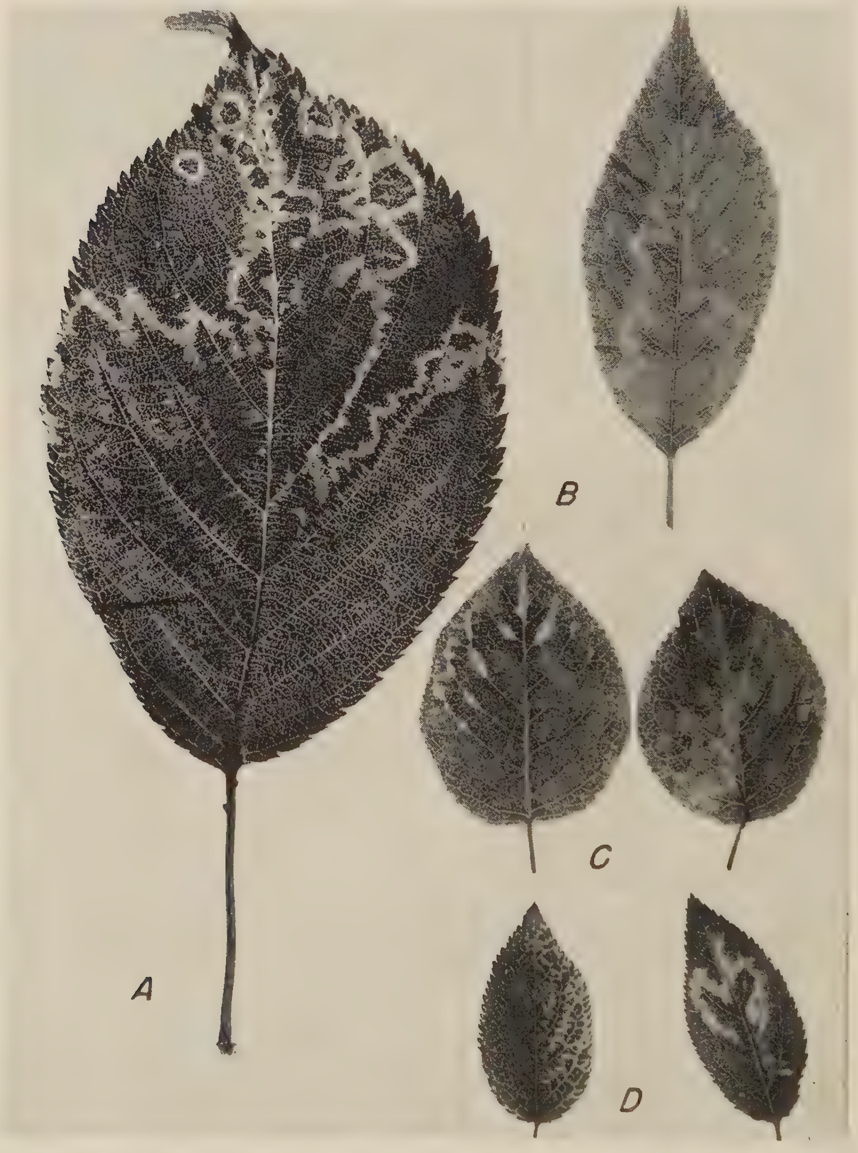

Figure 58.—*A*, Leaf of Amanogawa oriental flowering cherry affected with line pattern (banded chlorosis). *B–D*, Leaves of various species affected with the same disease: *B*, Montmorency cherry; *C*, mahaleb cherry; *D*, myrobalan plum.

Milbrath and Zeller found that the Michigan strain of the line pattern virus produced a pronounced golden blotch mottle with some ring spots, whereas the Oregon strain from Amanogawa oriental flowering cherry produced a banded oak-leaf pattern on some leaves and banded circular spots or irregular line patterns on others. The majority of the leaves showed no symptoms.

Sweet cherry.—On Napoleon and Black Tartarian cherries the virus caused similar symptoms, according to Willison and Berkeley. Symptoms were divided into two groups. The first comprised tiny fine rings, larger coarser rings, and faint oak-leaf patterns, which were more or less transient. The second group consisted of persistent watermark lines and more conspicuous yellow and later creamy-white lines up to 1 mm. in width. These lines formed striking irregular patterns, usually occupying less than a quarter of the leaf surface.

According to Zeller and Milbrath, on young foliage of Bing and Black Republican cherries line pattern virus caused faint line pattern symptoms which later became pronounced, whereas inoculations with buds from banded-chlorosis-affected trees caused well-formed, banded oak-leaf patterns on young foliage of Bing, Lambert, and Napoleon cherries. As these trees became older they showed chlorotic mottle.

Milbrath and Zeller found that mazzard cherry seedlings inoculated with buds from banded-chlorosis-affected trees vary greatly in symptom expression. Some seedlings showed a well-formed, banded oak-leaf pattern; others showed an irregular chlorotic or red mottling. In some seedlings tissues with these various patterns became necrotic and fell out, and then the leaves had a lacy appearance. The line pattern virus produced patterns similar in outline, but different in that the banding was of a bright-golden color.

Oriental flowering cherry.—On all affected varieties of oriental flowering cherries (Amanogawa, Shirotae, Naden, Kwanzan, and Temari) on which Zeller and Milbrath found the disease, the symptoms were similar (fig. 58, *A*). The borders were faintly chlorotic to pronounced golden or white. The patterns were sometimes made up of large rings, but more often they were of an oak-leaf type. The line pattern virus caused similar symptoms on Amanogawa, but the lines were less pronounced.

Diagnostic or Unusual Characteristics

Willison (*247*) considered as diagnostic for line pattern the striking yellow to white patterns on Shiro and myrobalan plums and Napoleon and Black Tartarian sweet cherries, the pale-green lines on peach, and the watermark lines on sour cherry. In comparing banded chlorosis with line pattern on several hosts, Zeller and Milbrath found the symptoms of banded chlorosis more pronounced than those of line pattern on the Amanogawa variety of flowering cherry and Bing and Black Republican sweet cherries and less pronounced on mahaleb and mazzard cherry seedlings and Abundance plum. Symptoms of the two diseases appeared identical on Shiro plum and J. H. Hale peach.

Of special interest is the effect of temperature on symptom expression, symptoms occurring only on leaves developed in the spring during cool weather. Differences in the varietal reaction of plums is also noteworthy, according to Willison and Berkeley.

Transmission

Transmission is easily effected by budding and bark-patch grafting. Evidence of natural dissemination was observed at East Lansing, Mich., (*47*) and in an orchard in Ontario by Willison and Berkeley. From these few observations there were indications that the vector is comparatively rare and the dispersion range is short.

Incubation Period

Trees budded in the summer produce affected foliage the following spring. Cation observed symptoms 6 weeks after inoculation under low-temperature conditions in the greenhouse in one trial in 1945.

Control Measures

It is doubtful whether roguing should be recommended. Use of disease-free stock and scion material for nursery propagation and top-working plum varieties is indicated by experiments of Willison and Berkeley and of Zeller and Milbrath.

Remarks

The combination of line pattern virus with the prune dwarf virus increases the virulence of prune dwarf in some hosts and of line pattern in others. The line pattern virus or close relatives seem to be widely distributed on stone fruits in various parts of the world (*247*). According to Zeller and Milbrath, banded chlorosis of oriental flowering cherry in western United States is closely related to, if not identical with, line pattern as found in the East on Japanese plum. The presence of this disease on ornamental oriental flowering cherry varieties indicates that it may have been imported from Japan, although as far as is known it has not been recognized in that country.

PLUM WHITE SPOT

By H. Earl Thomas and T. E. Rawlins

Other Common Names of the Disease

Santa Rosa plum mosaic.

Name of the Causal Virus

Aegrescit medendo Thomas and Rawlins.

Geographic Distribution

Plum white spot is known in Santa Cruz County, Calif., (*233*) and in orchards near Prosser, Wash. Distribution of this disease is probably wider, but the mosaics of plums are still to be distinguished clearly.

Economic Importance

This disease is probably of slight economic importance. The affected orchard first brought to attention of the authors was found to be more seriously damaged by other factors. Trees affected with plum white spot virus growing in good soil are not greatly impaired in vigor.

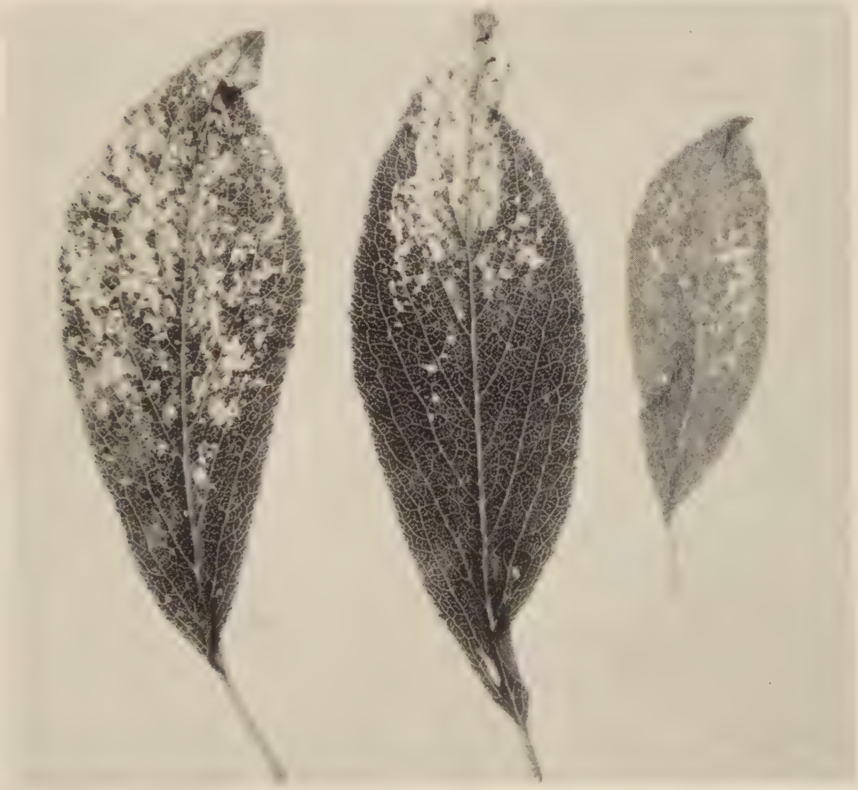

Figure 59.—Leaves of Santa Rosa plum naturally affected with plum white spot.

Host Range

Only the Santa Rosa plum (*Prunus salicina*) has been seen naturally affected, and other species have not been tested.

Symptoms

Symptoms consist of small, pale-yellow to paper-white spots tending to be aggregated toward the tip of the leaf (fig. 59).

Transmission

The disease is transmitted by grafting.

STANDARD PRUNE CONSTRICTING MOSAIC

By H. Earl Thomas and T. E. Rawlins

Other Common Names of the Disease

Standard prune mosaic.

Name of the Causal Virus

Vulnus immedicabile Thomas and Rawlins.

Geographic Distribution

Standard prune constricting mosaic is known in one orchard in Butte County, Calif., (*233*) and has been observed in one lot of nursery stock grown in central California.

Economic Importance

Eighty percent or more of the trees are infected in the one orchard known to be involved and the reduction in tree vigor seems to be considerable, but because of the limited known distribution the threat is potential rather than actual.

Host Range and Varietal Susceptibility

Standard prune (*Prunus domestica*) is naturally infected. Peach (*P. persica*) has been infected by inoculation. No symptoms developed on Sugar prune following inoculation by inarching with affected Standard prune.

Symptoms

Few to many chlorotic spots develop in the leaf blade of Standard prunes. These are often 1/16 to 1/8 inch in diameter and somewhat vague in outline. A distinctive feature is the concentration of the spots in a band across the tip

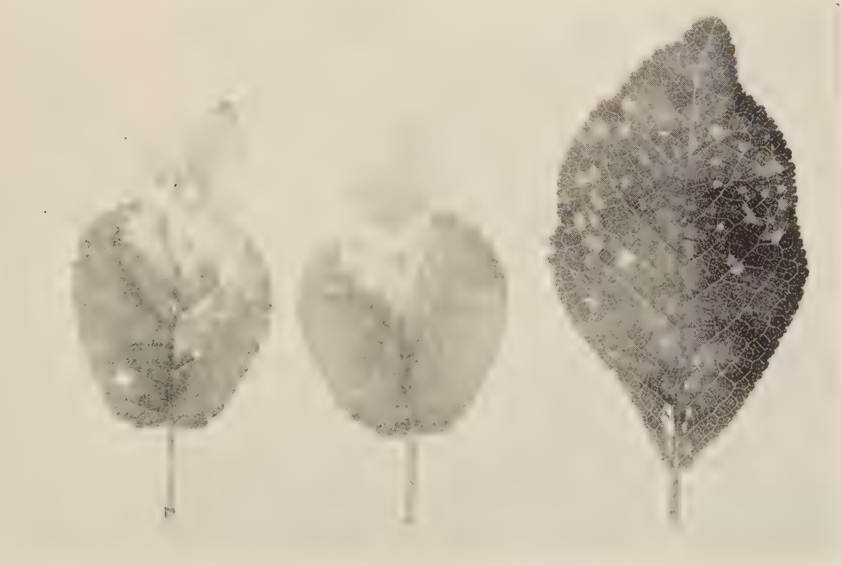

Figure 60.—Leaves of Standard prune affected with constricting mosaic.

half of the leaf blade; often necrosis follows, and all but the midvein in that area drops out (fig. 60). The symptoms are more conspicuous in warm weather than in cool. Symptoms on inoculated peach seedlings vary from indefinite mottling to clear-cut patterns of lines and rings. The damage to peach seems to be slight.

Transmission

Transmission has been effected by grafting.

Incubation Period

Plants inoculated in October and rested in midwinter developed symptoms in February of the following year.

Control Measures

The use of healthy buds of the prune for propagation of nursery stock is indicated.

RING POX OF APRICOT

By E. W. Bodine and E. L. Reeves

Other Common Names of the Disease

Ring pox has been called apricot ring spot in Colorado, and in Washington it is locally referred to as apricot pox and ring pox. Since the name "ring spot" was applied in 1941 to a virus disease of stone fruits affecting peach (*62*), the name "ring pox" proposed by Reeves (*184*) for this virus disease of apricots is preferred.

Name of the Causal Virus

Ring pox virus.

History and Geographic Distribution

Bodine and Kreutzer (*38*) observed this virus disease on apricots in Colorado in 1935, but the disease was described for the first time in 1942 under the name "ring spot" (*38, 40*). Reeves (*184*) noted its occurrence in Washington in 1939 and established its virus nature in 1941.

Economic Importance

Several hundred apricot trees affected with ring pox were removed in Mesa County, Colo., from 1936 to 1940. Since then, however, relatively few new cases have been observed. Ring pox has been reported from the Wenatchee and Yakima districts in Washington, but is considered of general minor importance in the State because of its low incidence. However, ring pox is viewed with concern since there has been evidence of field spread in both Yakima and Wenatchee Valley orchards. Most known diseased trees were removed from Washington orchards by 1945.

Host Range

So far as is known the virus of ring pox has been reported only on apricot (*Prunus armeniaca*). Orchard trees of the Montgamet, Wenatchee Moorpark, and Tilton varieties have been found affected.

Symptoms

Both the leaves and the fruits of affected trees exhibit symptoms of the disease. Reeves (*184*) reported some variation in symptoms on different varieties. In general the leaves of the Wenatchee Moorpark and Montgamet varieties develop irregular ring and angular spots and marked clearing of the principal veins, while those of the Tilton variety exhibit chlorotic areas. As the season progresses, the discolored tissues frequently become necrotic and crumble away, giving the leaves a ragged and shot-hole appearance. Under Colorado conditions developing fruits appear normal until approximately 2 weeks prior to ripening. After this time, the fruits exhibit protuberances, which often give individual fruits a bumpy appearance. During the ripening process the affected fruits fill out and tend to lose their bumpy appearance, but develop reddish-brown necrotic blotches, or ring spots (pl. 22). These discolorations normally extend into the flesh of the fruit. On ripe fruits with such symptoms cracks often develop in the discolored areas. On any thoroughly diseased limb all fruits show symptoms such as those just described. Under Washington conditions, recognizable fruit symptoms are apparent at least 6 weeks prior to harvest maturity and many of the fruits drop before they become ripe.

Transmission

Transmission has been effected through inoculation by bud and scion grafting only.

Incubation Period

Bodine and Kreutzer (*38*) reported an incubation period of approximately 2 years in the Montgamet variety. Reeves (*184*) inserted diseased buds into a 20-year-old Tilton tree in July 1941, and all fruits on the tree exhibited symptoms in May 1942.

Control Measures

Trees affected with ring pox are rendered commercially worthless and it is suggested that they be removed. Care should be taken in infected districts to ascertain that budwood sources are free of the disease.

VIRUS DISEASES OF ALMOND

ALMOND CALICO

By H. Earl Thomas and T. E. Rawlins

Name of the Causal Virus

Arcana caelestia Thomas and Rawlins.

Geographic Distribution

Calico is widely scattered and is probably general in many almond orchards in California, but there are at least four mosaic-type diseases of almond in the State still to be sorted out.

Economic Importance

No reduction in yield has been noted on trees showing well-defined leaf symptoms. The original tree from which the disease was described (*233*) was found to be affected also by another and more destructive virus.

Host Range

Natural infection has been found on almond (*Prunus amygdalus*). By inoculation, peach (*P. persica*) and mazzard cherry (*P. avium*) seedlings have been infected.

Symptoms

Almond.—Affected trees show variable amounts of foliage with chlorotic blotches in the leaf blade without pattern and with little distortion (fig. 61).

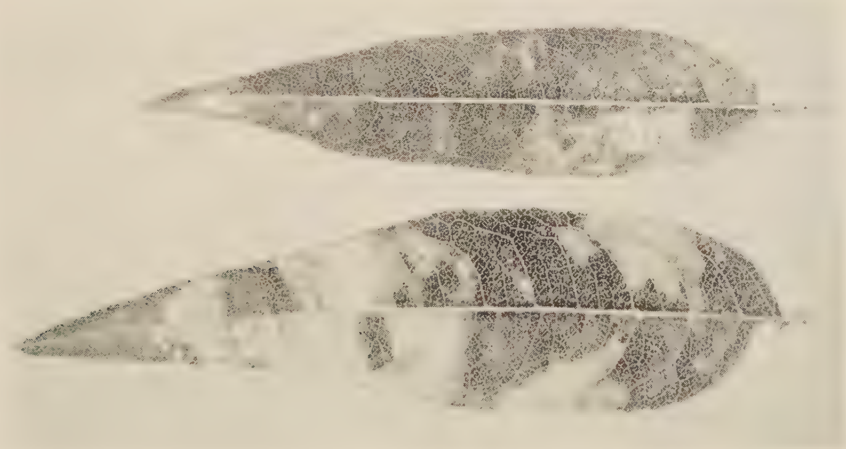

Figure 61.—Almond leaves affected with almond calico.

Peach.—Leaves on inoculated seedling peach trees slowly developed medium-large pale-yellow blotches without definite pattern or marked distortion.

Cherry.—Leaves on inoculated mazzard cherry seedlings produced chlorotic blotches or fairly definite lines with or without distortion; the symptoms were so variable as to suggest infection with a mixture of viruses.

Transmission

The disease is transmitted by grafting.

Incubation Period

Depending on the stage of growth of the host, the incubation period on almond varied from 7 weeks to 19 months.

DRAKE ALMOND BUD FAILURE

By E. E. Wilson and Gilbert L. Stout

Other Common Names of the Disease

None.

Name of the Causal Virus

Drake almond bud failure virus.

Geographic Distribution

So far the disease has been found only in three orchards located, respectively, in Yolo, Colusa, and Sacramento Counties, Calif.

Economic Importance

Because of its low incidence the disease causes negligible loss to the almond industry. In the one orchard where it is most prevalent, however, it has rendered a number of trees nonfruitful.

Host Range

The Drake variety of almond (*Prunus amygdalus*) is the only one so far found naturally affected. The Nonpareil variety is affected when artificially inoculated.

Symptoms

The failure of blossom and leaf buds to grow is the basic and characteristic symptom of the disease. All or most of the buds on a twig may fail to grow. When all buds fail, the twig remains alive during the spring but eventually dies. When the terminal bud is among those failing to grow, the twig dies back to the uppermost live lateral bud. Most or all lateral buds that grow develop into shoots rather than part into shoots and part into spurs (fig. 62). Thus, after a succession of bud failures, there is produced a limb with many branches, some of which are dead at the ends and none of which bear a full complement of leaves (fig. 63, *A*). Certain affected shoots taper somewhat more than normal ones, being thicker at the base but decreasing to an approximately normal diameter at the tip. The affected shoots not infrequently appear to be shorter than normal ones, and the leaves are more closely spaced.

Leaves on affected trees are slightly darker green and possibly somewhat larger than those on nonaffected trees. They appear to stand rigidly upright, not, as on nonaffected trees, bending away from the twig. Affected trees retain their foliage later in the fall than nonaffected ones. In fact some leaves may remain on the tips of the shoots until spring.

Affected trees blossom much less profusely than nonaffected ones, but the flowers are normal in shape, size, and color. The fruits, though normal in color, are few in number and frequently misshapen, the hulls being thicker than those on nonaffected trees and somewhat bumpy instead of smooth.

Transmission and Incubation Period

Drake almond bud failure has been transmitted to the Drake and Nonpareil varieties of almond by grafting scions from affected Drake trees onto nonaffected trees. Growth from the affected Drake scions showed symptoms of bud failure in 2 or 3 years. Severe bud failure developed on the inoculated

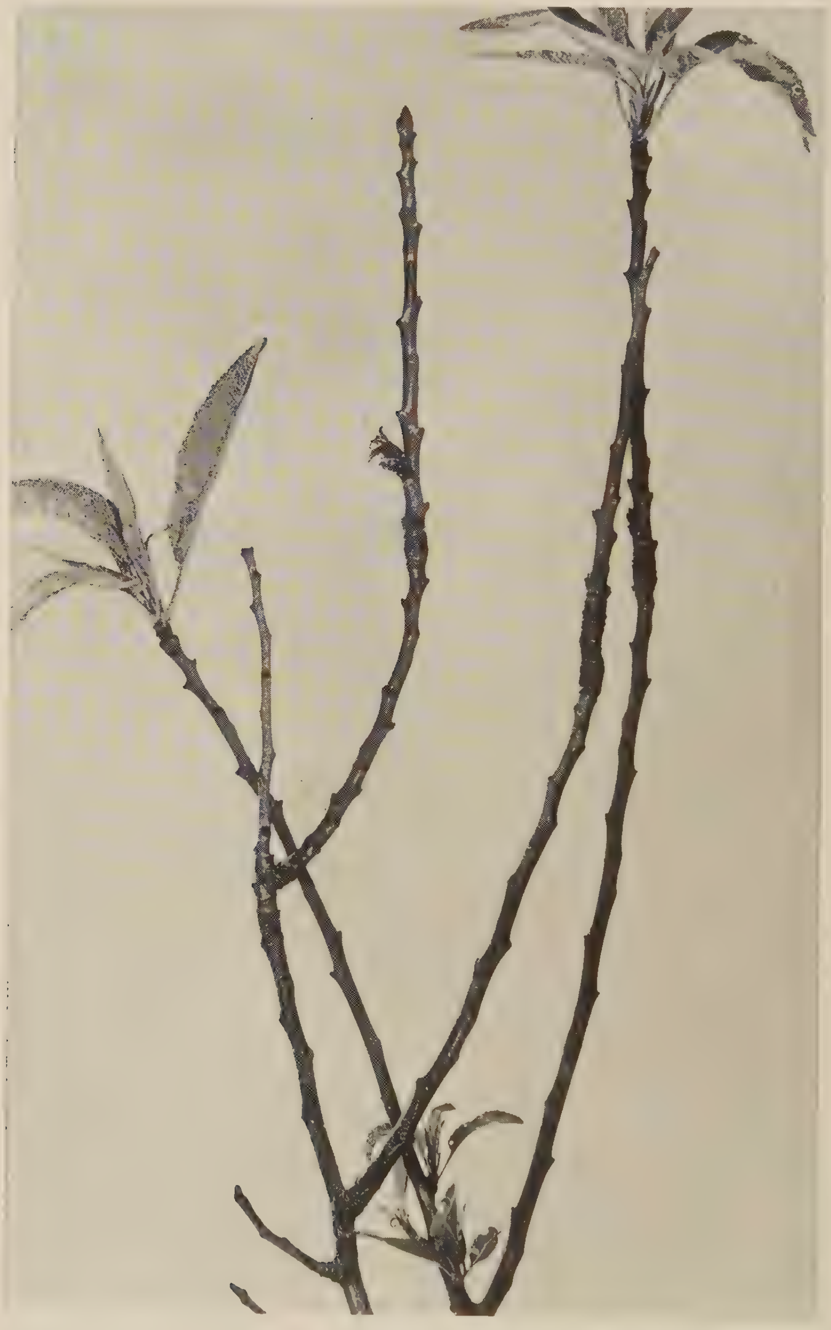

Figure 62.—End of a Drake almond branch, showing the effect of 3 years of bud failure. On the four shoots produced the previous year, only two terminal leaf buds, three lateral leaf buds, and one lateral blossom bud grew.

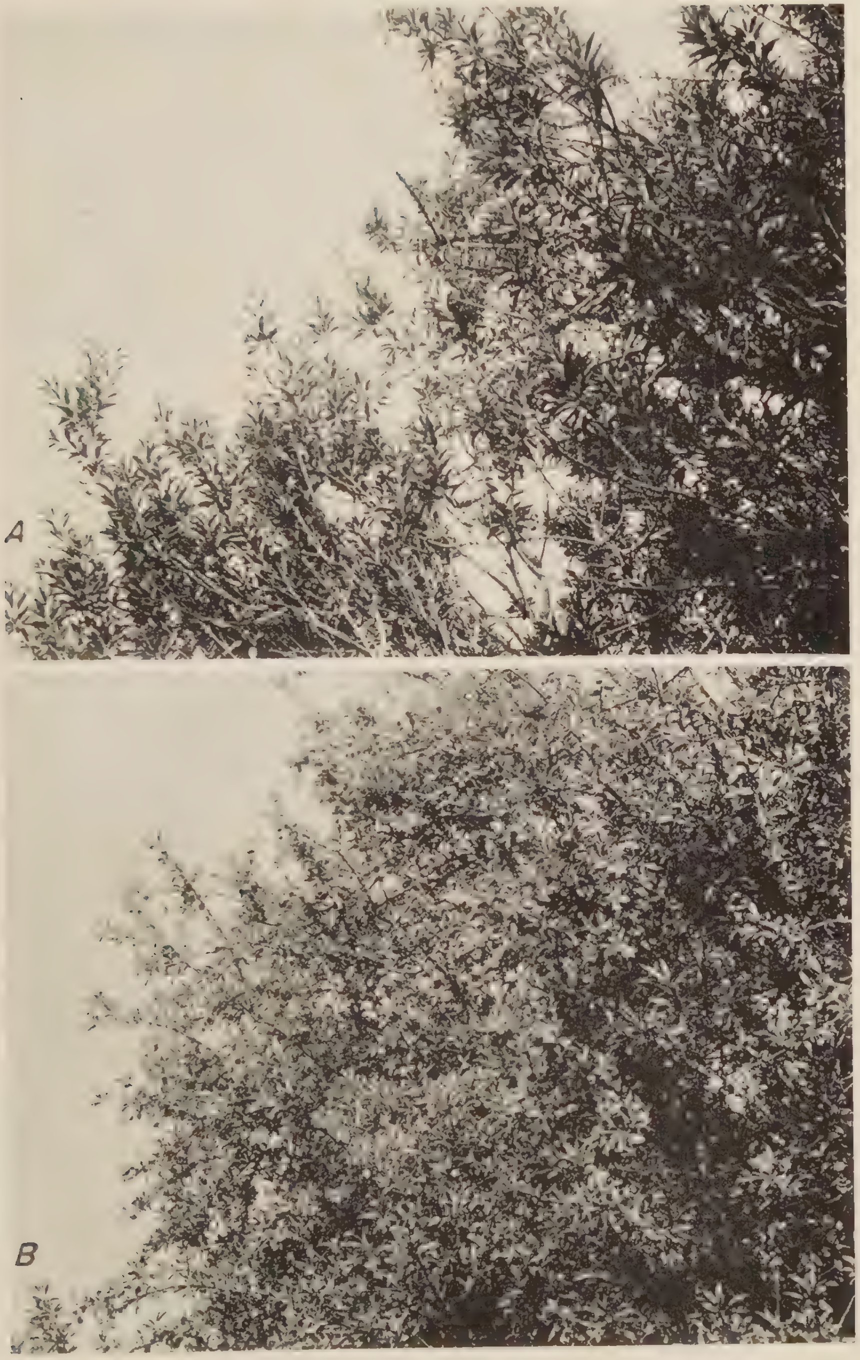

Figure 63.—*A*, Branches of a Drake almond tree affected with Drake almond bud failure showing rigid, upright leaves, bare twigs, and absence of fruit; *B*, comparable unaffected branches showing normal foliage and a good crop of fruit.

stock within 2 or 3 years. Within 4 years the trees were sparse in foliage and nonfruitful.

Remarks

A bud failure disorder with symptoms similar in certain respects to those on Drake occurs on Nonpareil, Peerless, and Jordanolo varieties in nature. Inasmuch as this disorder has not been transmitted by methods employed for the Drake disorder, it has been provisionally classified as nontransmissible. Formerly Drake almond bud failure was described with the nontransmissible almond bud failure (*229, 251*).

VIRUSLIKE DISEASES NOT PROVED TRANSMISSIBLE

SWEET CHERRY CRINKLE LEAF

By C. F. Kinman, W. B. Hewitt, T. B. Lott, G. L. Philp, E. L. Reeves,
R. S. Willison, and S. M. Zeller

Names of the Disease

The name "crinkle leaf" has become of such wide use among growers that, although it does not describe the disease adequately, it has been adopted. Among the names that have been applied to affected trees are "curly-leaf trees," "wild trees," "male trees," "red bud," and "unproductive cherry."

Causal Agent

The predominance of evidence indicates that crinkle leaf is not transmissible by the usual methods used for virus diseases and that it probably is due to a genetic bud variation.

History and Geographic Distribution

Crinkle leaf was first described by Kinman *(127)* under the name "unproductive cherry" in 1930. He reported its common occurrence on the Black Tartarian variety in many parts of California and Oregon. Subsequent reports and surveys showed the trouble to be so common on Black Tartarian that its known occurrence coincides with the distribution of this cherry variety in all the major sweet cherry sections of western United States and western Canada.

Economic Importance

Crinkle leaf is the cause of great monetary loss in orchards of affected varieties every year. Planting of affected nursery stock results in multiple loss including the original cost of the nursery trees and the cost of maintaining them in the orchard until they have been proved to be worthless. Surveys in Washington, reported by Coe *(66)*, showed that 4.1 percent of all the bearing sweet cherry trees in the State and 6.4 percent of the symptom-expressing varieties were affected with either crinkle leaf or deep suture. Bing and Black Tartarian are the chief commercial varieties affected. These are important varieties in the sections of western United States and southwestern Canada where the production of sweet cherries is an important industry; thus in these sections there is a correspondingly higher percentage of crinkle leaf in cherry orchards. In Washington some growers have planted Napoleon (Royal Ann) and Lambert in place of Bing and Black Tartarian, in order to avoid crinkle leaf.

Suscept Range and Varietal Reaction

The list of affected varieties is based entirely upon the occurrence of symptoms; since the disorder is apparently noninfectious, there is no way of determining the absolute relationship of the symptoms on the various varieties. Crinkle leaf has been reported on sweet cherry *(Prunus avium)*, and similar symptoms have been seen by Reeves and Cochran [43] on certain varieties of domestica plum *(P. domestica)*, notably the Italian Prune. Reeves *(184)* reported 1.1 to 1.3 percent of crinkle leaf on mazzard (*P. avium*) seedlings. Crinkle leaf is most common on the Black Tartarian and Bing varieties of sweet cherry. It has been seen also on Black Republican, Eagle, Burbank, Dr. Flynn, Ox Heart (of America), Shelton, and Waterloo Heart varieties. Kinman (*127*) reported the occurrence of crinkle leaf on Lambert cherry, but Reeves (*184*) stated that it has not been seen on Lambert or Royal Ann (Napoleon). It is possible that crinkle leaf would have been as prevalent on other symptom-expressing varieties as on Black Tartarian and Bing had they been as widely planted.

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[43] Reeves, E. L., and Cochran, L. C. Unpublished data.

Symptoms

Leaves on affected trees are variously misshapen and mottled (figs. 64 *A*, *B*, and 65, *C*); margins are often indented and abnormally serrated through failure of blade tissue to develop. Some leaves are oddly and severely distorted with deep sinuses and lobes and accentuated tips and bases. If removed from the tree, they would not be recognized as cherry leaves. The inhibited areas are usually lighter green and have a silvery upper surface and many more veins per unit area than the remainder of the leaf, which is darker green than normal. The light-green areas are present when the leaf first appears in the spring. Uneven expansion appears to be associated with a mixture of tissues some of which have more chlorophyll than others. Leaves developed later in the growing season, especially on sucker shoots, are often less affected than those produced in the spring. The total leaf surface of a moderately affected tree is only approximately three-fourths that of a normal tree; yet affected trees are not noticeably reduced in growth.

The unfruitfulness of trees affected with crinkle leaf is not due to failure to blossom, but many of the blossoms are smaller than normal and are visibly defective. The peduncles are short, and most pistils are short and slender and have a tendency to discolor early (fig. 65, *A*). Some of the discolored pistils are brown before the flowers open, and others become brown soon afterward; and few develop into fruits. Such fruits are small, pointed, and often with a raised suture (fig. 65, *C*); they are attached with their long axis at an angle to the stem. Flowers near the base of the preceding season's growth often fail to open fully. The swollen buds on such growth become reddish, and some of them remain in place for a number of weeks.

In many instances a tree may appear entirely normal, but upon close examination small branches, twigs, or even a few leaves can be found to be affected. It is common for trees to have both normal-appearing branches and those affected with crinkle leaf. In some instances an entire tree is badly affected, and in others one or more branches may be severely affected and others on the same tree bear normal leaves and fruits.

Transmission and Spread

Kinman (*127*), working with Black Tartarian cherry, showed that crinkle leaf can be perpetuated through buds but is not transmitted either when affected trees are top-worked with normal scions or when affected scions are placed on normal trees. Zeller and Evans (*259*), working with Bing cherry, reported transmission of a vein-clearing disease that is very similar to crinkle leaf if not identical with it. Failing to repeat transmission in later work, Zeller and Milbrath[44] concluded that the inoculated test trees for which transmission was reported may have had an inherent form of crinkle leaf that was not apparent when the inoculations were made. This conclusion is supported by the development of crinkle leaf in nursery trees propagated from apparently normal trees that later were found to have a few spurs affected with crinkle leaf. The orchard incidence of crinkle leaf, its occurrence in

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[44] Zeller, S. M., and Milbrath, J. A. Unpublished data.

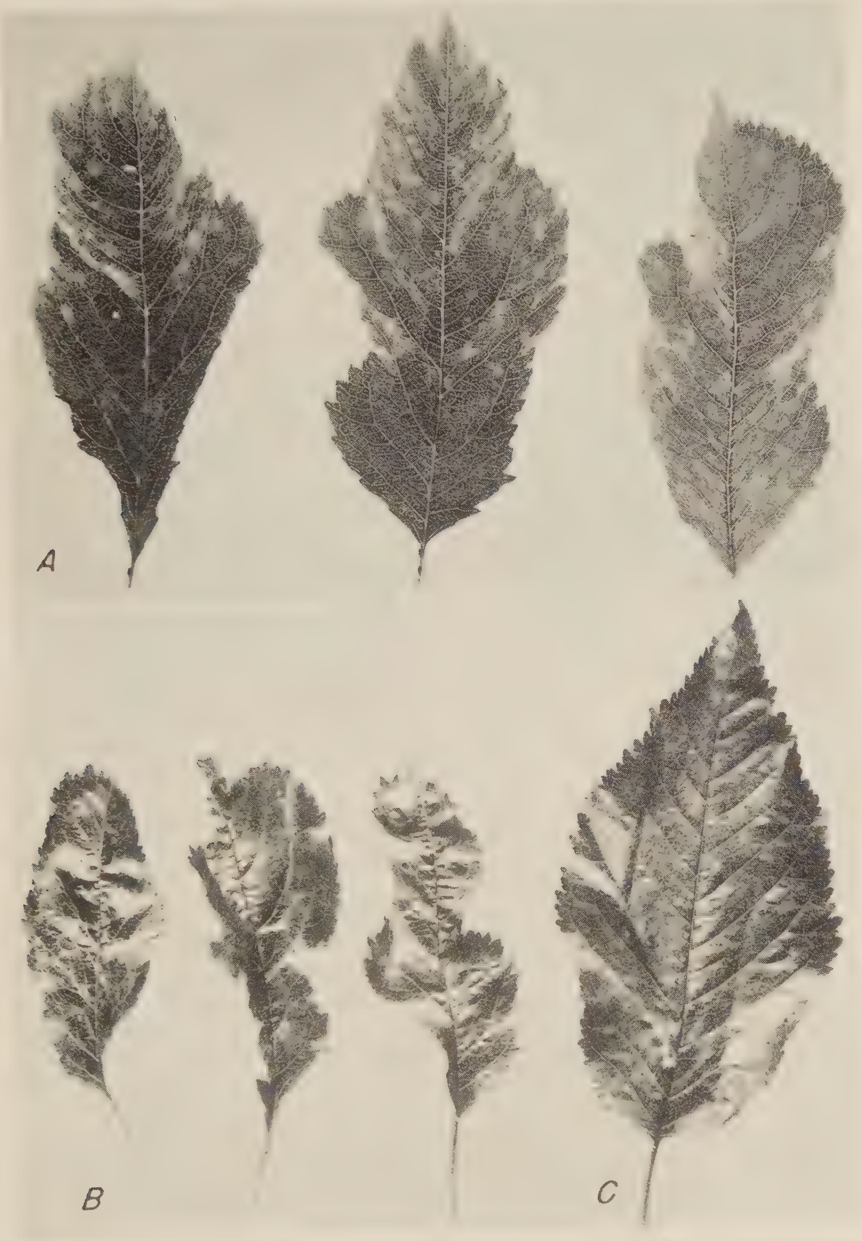

Figure 64.—Sweet cherry leaves affected with crinkle leaf: *A*, Bing; *B*, Black Tartarian. *C*, Comparable leaf from an unaffected Black Tartarian cherry tree.

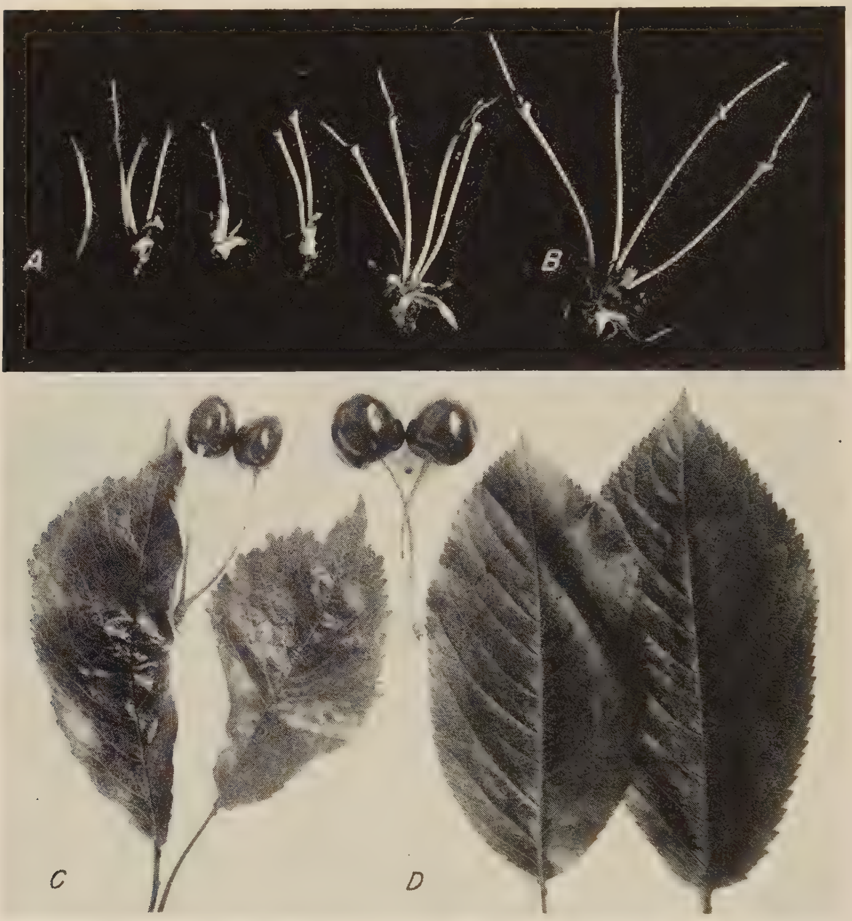

Figure 65.—*A*, Exposed pistils of blossom clusters from Black Tartarian cherry affected with crinkle leaf; *B*, comparable pistils from an unaffected tree; *C*, fruits and leaves from a Burbank cherry affected with crinkle leaf; *D*, comparable fruits and leaves from an unaffected tree.

portions of trees developed from a single bud, and the common impression and circumstantial evidence that crinkle leaf is spreading in individual trees as well as in commercial orchards suggest to some investigators a transmissible agent. Since growths from normal scions placed on trees affected with crinkle leaf have remained normal for several years and thus without evidence of transmission, it is difficult to explain crinkle leaf on a virus hypothesis without a new concept.

Careful observation is often required to detect traces of mild symptoms of crinkle leaf, and these symptoms are often overlooked when a propagating wood is selected. It is probable that the disease in many cases has been spread in nursery stock propagated from such trees.

Hewitt and Philp found that some of the hybrid seedlings developed crinkle leaf when the pollen parent was affected with crinkle leaf and the seed parent was normal. They noted crinkle leaf also on seedlings of which the seed parent was affected and the pollen parent was normal. Reeves (*184*) reported the occurrence of occasional crinkle-leaf-affected seedlings among those grown from certain commercial lots of mazzard seeds.

Control Measures

Control of crinkle leaf is a matter of avoidance and replacement. Crinkle-leaf-affected trees are common in the stock of many commercial nurseries. This is undoubtedly due to lack of knowledge of the malady, insufficient care in selecting budwood, or selection of budwood in the fall when crinkle leaf is less easily detected than in spring and summer. Budwood sources should be selected in the spring before the fruit is mature. No budwood should be taken from a tree showing any crinkle-leaf-affected leaves. There is definite need of a budwood-certification program and the prospect of success in control is good if certified budwood is used. Affected trees may be top-worked with good scions. It is doubtful whether it would be worth while to top-work old trees, but top-worked trees up to 6 or 8 years old would fruit sooner than replants.

Remarks

Kinman (*127*) suggested that crinkle leaf is similar to the condition found in variegated plants on which occasional normal leaves, twigs, and branches occur. The rather common occurrence of variegated twigs on normal plants might parallel the frequent occurrence of crinkle leaf twigs and branches on otherwise normal cherry trees. When larger portions of a tree are crinkled, the mutation might have occurred earlier in the life of the tree and might have been perpetuated in succeeding growth. To support this hypothesis, it must be assumed that a cherry variety like Black Tartarian or even some clones of Black Tartarian are inherently constituted to mutate whereas others are not.

Willison suggested that some strains of Bing, Black Tartarian, and certain other varieties possess genetic weaknesses to a greater degree than do other strains and that in such strains sporting in the direction of crinkle leaf may occur frequently. Spread of the disease could occur in this manner if such strains were used in propagation even if they were apparently normal when first set out in the orchard.

Milbrath and Zeller[45] opposed the bud-sport concept as an explanation of crinkle leaf. Their observations on crinkle leaf gave more support to the disease concept. It is difficult, they claimed, to conceive of two or more varieties such as Bing and Black Tartarian mutating such identical bud sports or of a bud sport remaining latent for several years and then cropping out in a single branch or in an entire tree. Crinkle leaf in the Bing variety especially is difficult to explain on the hypothesis of mutation. The Bing variety is a seedling of *P. avium*, and genetically all vegetative portions of it should be identical. But in reality it is found that some subsequent propagations of this one original tree develop a tendency to manifest crinkle leaf consistently, while other lines of propagations from the same tree never produce crinkle leaf.

Kinman, observing crinkle-leaf-affected trees in California, found that the number of crinkle leaf shoots are more numerous on old wood of trees that, because of age, drought, or neglect, are low in vigor than on trees that produce good terminal-shoot growth. Trees somewhat low in vigor produce a greater number of small lateral shoots along the part of the branch that is old than do vigorous trees, and it is among these shoots that the occurrence of crinkle leaf is most frequent. The frequency with which lateral crinkle leaf shoots develop on affected trees gives the impression that the trouble is spreading as the tree grows older rather than making more frequent appearance on trees already affected. Seldom is a new crinkle leaf terminal shoot found on a tree producing vigorous terminal shoots. But few lateral shoots are produced by such trees along the part of the branch that is a few years old.

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[45] Milbrath, J. A., and Zeller, S. M. Unpublished data.

SWEET CHERRY DEEP SUTURE

By E. L. Reeves, Earle C. Blodgett, C. F. Kinman, T. B. Lott,
J. A. Milbrath, and S. M. Zeller

Names of the Disease

The name "deep suture" has become generally accepted by growers and research workers although it applies only to the fruit symptom and makes no reference to the accompanying leaf distortion. Other names which have been applied are "long leaf" and "rough leaf." The name "rough leaf" was first employed by Kinman in his early unpublished field records on the disease.

Causal Agent

Information concerning the cause of deep suture is incomplete. It has been considered as possibly being a frequently occurring mutation. Deep suture has not proved to be transmissible by the usual methods used to transmit virus diseases although the unusual malformation of both fruits and foliage along with the sporadic orchard occurrence might possibly suggest the presence of a transmissible entity.

History and Geographic Distribution

Deep suture was first described by Reeves (*183*) as a disorder sometimes confused with mottle leaf. Kinman observed the condition he referred to as rough leaf in Sacramento Valley, Calif., cherry orchards during the late 1920's. The condition has been reported on trees in California, Idaho, Montana, Oregon, Utah, and Washington and in British Columbia, Canada, but since its occurrence is common on some named varieties and mazzard seedlings, it is suggested that deep suture is probably widespread.

Economic Importance

It is difficult to estimate the economic losses from deep suture because the severity on affected trees varies and the degree of fruit malformation often varies with the growing season. Deep suture has been reported as causing moderate loss to Washington cherry growers, but it is not considered to be so serious as crinkle leaf (*184*). Coe (*66*) grouped deep suture and crinkle leaf together for purposes of the survey in Washington, and he stated that it was not unusual to observe symptoms of both disorders on the same tree. He reported that less than 10 percent of the properties inspected were found free of the disorders.

In 1937 Kinman examined the Bing trees in three orchards in Sacramento Valley and found that 13 percent of the trees observed were very severely affected with deep suture and that more than 50 percent had a trace of the condition.

In Oregon many of the Bing trees are affected, and it has been difficult to find trees absolutely free of deep suture from which to obtain propagation wood. In some districts fruit of affected trees is not picked because packers will not accept it.

Crop loss from deep suture varies, because only parts of some trees are affected and the portion of the fruits that are malformed even on affected arms varies from season to season. Use of affected nursery stock results in a multiple loss composed of the cost of the nursery tree plus the cost of maintenance in the orchard until the tree is proved worthless.

Suscept Range

Since deep suture has not yet been proved infectious, the determination of affected plants is based entirely upon visual symptoms. Deep suture has been reported only on sweet cherry (*Prunus avium*). Reeves (*183*) stated that the disorder was found in 1938 on Bing and Lambert cherry trees, but it is now questionable whether two affected trees supposedly Lambert were truly that variety. Bing is the principal variety found affected, but occasional Black Tartarian trees and mazzard (*P. avium*) seedlings exhibit symptoms. One tree thought to be the Black Republican variety was observed affected, and no doubt other varieties that exhibit symptoms may be found.

Symptoms

Bing cherry trees affected with deep suture have variable percentages of malformed leaves (fig. 66) and fruits, and different trees display different degrees of malformation. Sometimes entire trees are severely affected, but at other times branches scattered through a tree or only certain lateral ones

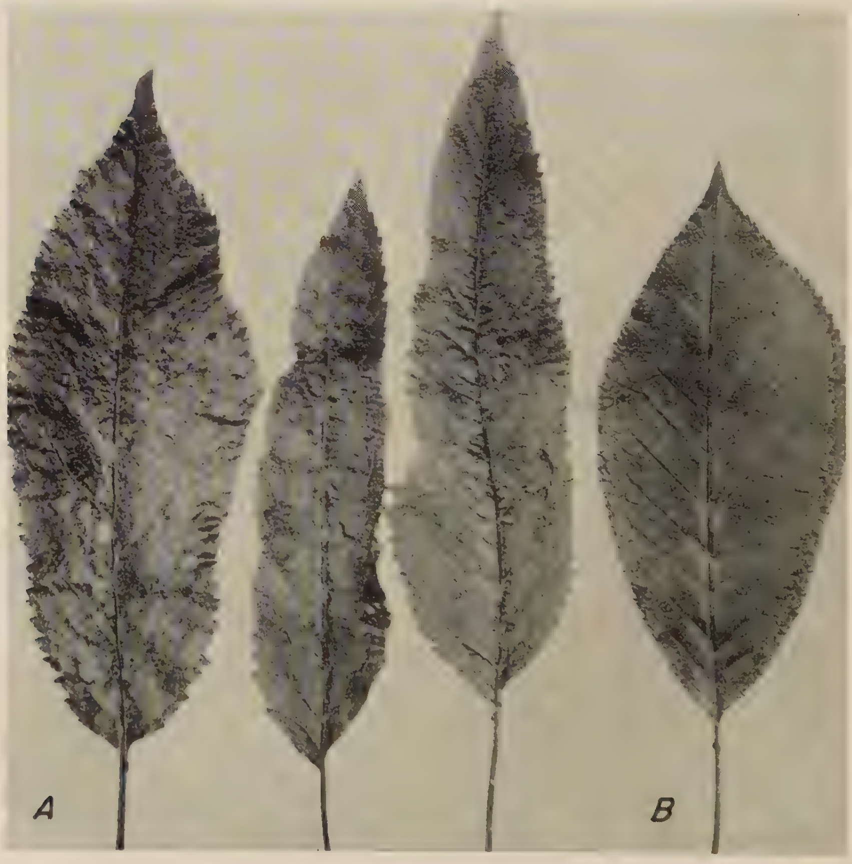

Figure 66.—*A*, Leaves from a Bing cherry tree affected with deep suture; *B*, comparable unaffected leaf.

exhibit symptoms. A severely affected tree viewed from a distance often has a drooping appearance like one suffering from drought. Nursery trees affected with deep suture are often greatly dwarfed in height. When such trees are transplanted, they grow very slowly and the foliage is sparse and malformed.

Some trees show very little leaf malformation other than a thicker, more leathery texture and a slight rugosity, while the leaves of others are reduced to narrow ribbons, many of which may be only a midrib in parts and not over 1 inch wide in other parts. The mildly affected trees may show the characteristic deep suture type of leaves in the spring, but the leaves produced in summer may be nearly normal in shape.

Some leaves are often actually longer than normal and their lateral veins are shorter on one side or both sides of the midvein, whereas other leaves merely appear to be longer because of their relative narrowness. The surface of the leaf may be very rough, but not puckered. The periphery of some leaves may be irregular, and perforations occur on a few. The leaf margins usually have fewer serrations than normal, sometimes none in portions. A typical leaf from a Bing cherry tree affected with deep suture is shown in plate 19, *bottom*. A faint interveinal chlorosis may be present on some leaves during the late spring, but it is less evident later in the season. Pronounced chlorotic areas like those found on trees affected with crinkle leaf do not occur. Affected leaves often tend to develop a bright sheen on the surface and are darker green than normal. Late-spring frosts sometimes cause certain leaf malformations to develop on normal leaves; some of these may easily be confused with the leaf symptoms of deep suture.

Affected fruits have a pronounced depression on the suture side and are normally rounded at the end, as shown in plate 19, *bottom*. Fruits with pronounced deep suture are most often borne relatively near leaves that exhibit symptoms.

Transmission

While fewer transmission experiments have been attempted with deep suture than with crinkle leaf and over a shorter period, the two disorders have behaved the same in all transmission tests thus far made. Zeller and Milbrath, working in Oregon, and Reeves, working in Washington, made numerous unsuccessful attempts to transmit deep suture. Growth from normal scions grafted on trees affected with deep suture was normal, and scions from affected trees grafted on normal trees produced affected growth but failed to transmit the disorder to the stock.

Deep suture has been observed affecting mazzard seedlings, although there are no available data on the percentage of seedlings that might be so affected. This could be interpreted as transmission of the disease through the seed, but further evidence would be needed to substantiate such an interpretation.

Just as with crinkle leaf, there is considerable circumstantial evidence that deep suture is spreading in commercial orchards; this spread suggests a transmissible agent. As far as reported, the possibilities of transmitting deep suture have been inadequately explored.

Control Measures

Control is considered at present to be largely a matter of prevention, and the careful selection of nonaffected propagation wood is especially recommended as a preventive measure. Scion wood should preferably be taken from large bearing trees that have no trace of the disorder; and selection of budwood from blocks where deep suture does not occur would be even more desirable. It would be very desirable if nurserymen had access to established "mother blocks" of trees free from the disorder and could take their propagation material from these. Budwood sources should be selected just before fruit maturity.

Remarks

Zeller and Milbrath made the following observations: Buds from the normal portion of a Bing tree with only one isolated branch affected with deep suture near the top consistently produced both normal and deep suture trees. Some showed deep suture foliage in the nursery row, and even though they appeared normal in the nursery row others developed deep suture the first year after transplanting and still others developed deep suture after 3 or 4 years. It is difficult to accept the hypothesis that this disorder has the nature of a frequently occurring mutation, especially when a tree develops normally for a while and later shows the disorder. The ability of deep suture to remain latent points more to a virus behavior. Furthermore, it is difficult to conceive why one Bing tree should remain normal, whereas another has the ability to mutate freely and repeatedly to produce deep suture when the Bing variety supposedly is the progeny of a single seedling of *Prunus avium.*

Since Reeves found that growth from normal scions grafted on trees affected with deep suture remained normal after 8 years and scions from affected trees grafted on normal trees did not transmit the disorder, it is difficult to explain deep suture on a virus hypothesis without a new concept.

ALMOND BUD FAILURE

By E. E. Wilson and Gilbert L. Stout

Other Common Names of the Disease

Shatter bud, crazy top, and mule tail.

Causal Agent

Unknown.

Geographic Distribution

Trees affected with almond bud failure occur in orchards scattered through the interior valleys and coastal districts of north-central California.

Economic Importance

Almond bud failure is of minor importance except in certain orchards in which a high percentage of the trees of certain varieties develop the disorder while young. On such trees the yield is variously reduced, some trees producing only a few nuts.

Suscept Range

Nonpareil, Peerless, and Jordanolo varieties of almond (*Prunus amygdalus*) are the only ones known to be affected.

Symptoms

The chief feature of the disease on all varieties is the failure of many of the leaf and blossom buds to live and develop. On some twigs all buds grow, but on others only a few buds do so. Twigs on which all buds fail retain life during the spring, but eventually they die. Buds that grow produce shoots rather than spurs. Such shoots appear normal the year they are produced, but many of their buds fail to grow the following year.

Though the disorder commonly occurs on all branches of affected trees, sometimes it is confined to one or two branches. Occasionally all but one or two branches of an affected tree show symptoms.

Elongation of affected limbs is more or less normal, but some twigs do not attain normal diameter and in consequence are slender and willowy (fig. 67, *A*).

When the terminal bud is among those dying, shoot growth is produced from other buds. The shoots thus formed are often at various and peculiar angles with the branches from which they arise. A succession of bud failures results in a branch which is crooked (frequently growing back on itself, especially in Peerless), sparse in foliage, and bearing deranged, slender, willowy twigs more or less clustered at certain places (*229*, *251*). In Peerless particularly, brown necrotic areas often develop on the bark of twigs produced the previous season. These areas later become rough and cracked, forming bands of rough bark up to a foot long on older wood.

In all varieties leaves and blossoms on affected branches, though fewer than on nonaffected branches, are normal in size, shape, and color. A delay in the opening of blossoms, however, is common on severely affected trees.

Transmission

Up to the present time the disorder of the Nonpareil and Peerless varieties has not been transmitted to either of these varieties, though it has been bud-perpetuated from both. Nonaffected Nonpareil and Peerless trees grafted

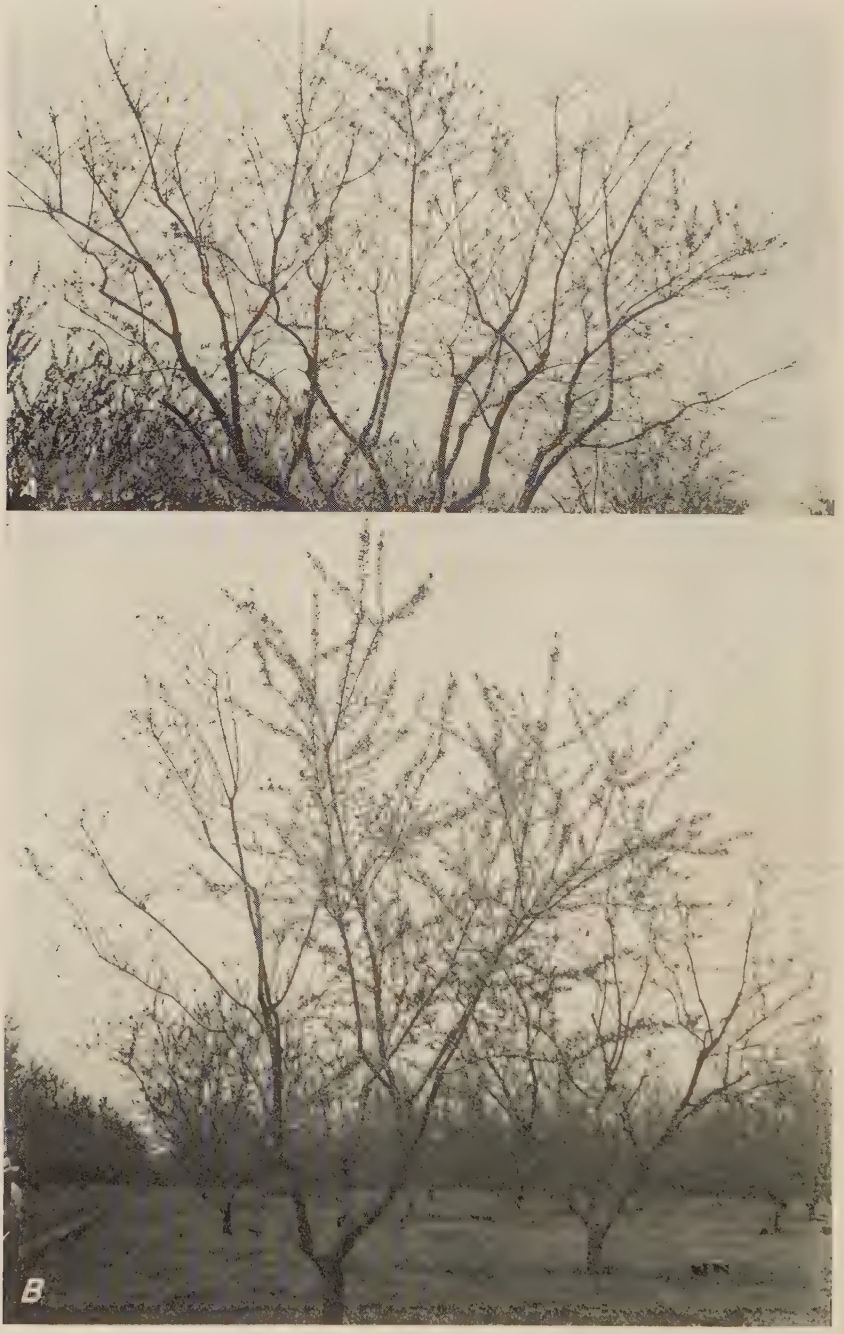

Figure 67.—*A*, Nonpareil almond affected with almond bud failure, showing in center one vigorously growing branch less affected than the others; *B*, Nonpareil almond tree showing at right unaffected branches that developed from a healthy graft placed on understock 7 years previously and at the left the branches that developed from the affected understock.

with scions from affected Nonpareil and Peerless trees have remained without symptoms of bud failure for 6 years, though about 80 percent of the branches produced from the affected scions developed symptoms within 3 years. Moreover, nonaffected Nonpareil scions grafted onto affected Nonpareil trees have remained symptomless for 11 years.

In figure 67, *B*, is shown such an almond tree grafted 7 years previously. In another series growth from buds taken from affected trees and placed on seedling peach and bitter almond developed bud failure symptoms within 3 years.

Inasmuch as the disorder was found in the Jordanolo variety only recently, transmission tests with this variety are not yet complete.

In general, the symptoms of the disorder on Nonpareil, Peerless, and Jordanolo almonds resemble those of the transmissible bud failure of Drake almond described on page 191. The failure of buds to grow is a basic and characteristic symptom of both diseases. The symptoms of almond bud failure on naturally affected Nonpareil almond are similar to those produced on this variety as a result of inoculation with scions from Drake trees affected with Drake almond bud failure. The symptoms of almond bud failure on Nonpareil, Peerless, and Jordanolo varieties differ from those on the Drake variety affected with Drake almond bud failure as follows. (1) Affected twigs are slender and willowy instead of normal in diameter, and they arise at various and peculiar angles instead of normal ones. (2) Leaves are normal in size, color, and shape and are shed at the normal time instead of being larger and slightly darker green than normal and persisting late in the fall, sometimes even until spring. (3) The fruits are normal in shape instead of being somewhat malformed.

Judging from the result of transmission studies the malady of Drake almond may not be the same as that of the other three varieties. Differences in certain details of symptoms would seem to confirm this view. Inasmuch as not all possible methods of transmission have yet been exhausted, the authors regard the placing of the Nonpareil, Peerless, and Jordanolo disorder in a nontransmissible classification as provisional only.

NONINFECTIOUS PLUM SHOT HOLE

By C. O. Smith and L. C. Cochran

Name of the Disease

The name "shot hole" has been variously applied to both infectious and noninfectious plant diseases. The disease described in this section is of the latter type, is genetic as evidenced by perpetuation in buds, and is heritable as evidenced by passage through seeds.

Geographic Distribution

The disease has been seen rarely in native thickets on seedlings of wildgoose, or pottawattamie, plum (*Prunus munsoniana*) in southwestern United States. Many selections have been made from seedlings of *P. munsoniana* and are widely grown as dooryard trees and in small plantings in southern United States. The most common of these have bright-red fruits and are called wildgoose. Shot hole of various types has been seen on individual trees scattered through the area.

Economic Importance

Noninfectious plum shot hole is not of commercial importance, but it may cause confusion when it is associated with infectious diseases.

Suscept Range

Noninfectious plum shot hole (*205*) was originally described on Beaty plum, which, according to Wight (*245*), is a hybrid between *P. angustifolia varians* and *P. munsoniana*. Individual plum seedlings from seed collected from open-pollinated pottawattamie plum collected in Utah were variously affected, symptoms ranging from purple spots to large necrotic spots and shot holes indicating genetic segregation. It seems logical that the trouble may be the result of a genetic weakness of *P. munsoniana* and that it should be expected in plums arising as seedlings and hybrids of this species.

Symptoms

Early-spring growth of affected trees appears normal. As the season progresses, the older leaves at the base of the shoots develop translucent flecks, which rapidly enlarge. The centers of these spots turn brown (fig. 68, *A*), and in the majority of cases the brown areas separate from the rest of the leaf at a definite line of cleavage and drop out. The resultant shot-hole condition develops progressively toward the apex of the shoots, and by midsummer, when growth has become slower, the foliage appears riddled with holes (fig. 68, *B, C*). The spots and holes on leaves of most trees range from pin-point size to ⅛- to ¼-inch diameter, the size usually being inversely proportional to the number on the affected leaf. Individual affected seedlings vary greatly in symptom expression, some being severely and others only mildly affected. On some the spots are all small and appear as purple dots, or freckles, and the centers do not fall out. On others the spots are all large, and the centers drop out as they are formed. The spots are usually circular, but the larger spots may be irregular in shape, especially where two or more spots run together or occur in the angles of veins.

Transmission

When buds or scions taken from diseased plants are placed on unaffected sister seedlings or on any of several horticultural plum varieties and on other

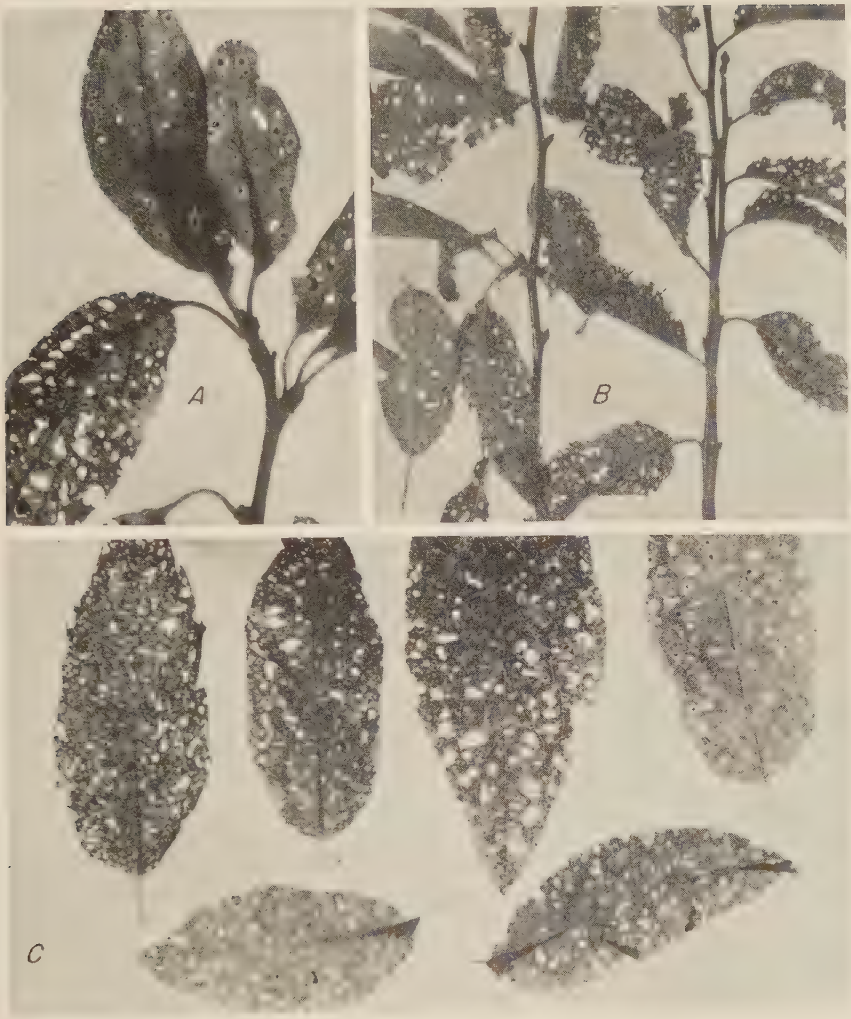

Figure 68.—Beaty plum seedling affected with noninfectious plum shot hole: *A*, Early stage, showing zones around dark centers; *B* and *C*, advanced stages.

species of *Prunus* the shot-hole symptoms develop on the leaves of shoots arising from the affected bud, but in no case have they been seen on the understock below the point of grafting. It can be argued that failure of transmission is due to nonsusceptibility, but until there is some evidence of transmission it seems most logical to assume that this disease belongs to the bud-perpetuated, nontransmissible group.

Remarks

Noninfectious plum shot hole described here appears to be connected with *P. munsoniana.* The trouble occurs on certain other plums of the wildgoose type, most of which have been selected from *P. munsoniana,* and should be expected when this plum is used as a parent. No connection has been established with Italian Prune leaf spot as described by Blodgett (*16*), but the two diseases have characters in common and may be similar.

ITALIAN PRUNE LEAF SPOT

By Earle C. Blodgett and B. L. Richards

Other Common Names of the Disease

Italian Prune leaf spot is sometimes called shot hole.

Geographic Distribution

Leaf spot is generally found wherever the Italian Prune variety is grown in Idaho, particularly in the southwestern part. It occurs in the Milton–Freewater and The Dalles districts of Oregon; in Yakima Valley, at Walla Walla, and in Wenatchee Valley, Wash., and wherever the Italian Prune is grown in Utah. Leaf spot apparently occurs in the Midwest, because affected nursery stock has been received from an Iowa nursery, and possibly it occurs farther east. It is, of course, possible that the Iowa nursery stock was propagated in the West or that scion wood from western sources was sent east.

What appears to be a disease belonging to the same class was described by Smith and Cochran (*205*) on Beaty plum in California.

Economic Importance

The actual loss from Italian Prune leaf spot is impossible to estimate accurately, but the disease is regarded as the cause of serious losses in quantity and quality of Italian Prune fruit. Losses vary with season, indicating an effect of environmental factors during the growing period. It appears from surveys in Idaho that in general the percentage of affected trees is greater in young orchards than in old plantings. Richards observed in Utah that the older orchards seem to be more severely affected and that Italian Prune production is on the way out, principally because of leaf spot.

Suscept Range

The Italian Prune variety (*Prunus domestica*) is the only host considered here, although Smith and Cochran (*205*) described a similar type of disorder on Beaty plum in California. Blodgett observed perpetuation of normal leaves on growth from scions of an unidentified prune of the Italian Prune type grafted on severely affected Italian Prune stock.

Symptoms

The symptoms of Italian Prune leaf spot apparently do not appear until early summer, as the first growth seems to be normal. The spots on the leaves vary in size from very small (1 to 2 mm.) to large, round or irregular blotches of dead tissue (pl. 18, *B*). Sometimes the leaf margins die and dry up. The affected tissue is brown and has definite margins, and there are often distinct concentric zones in the spots. Frequently the spots drop out, leaving a shot-hole condition. A leaf mottle frequently precedes or accompanies the necrotic spotting (*22*). This condition is widely prevalent and is characterized by light-green or rusty areas in the leaves. The mottling may be very indistinct or very striking, much like mosaic symptoms. Mottling may be closely associated with leaf spot, or it may occur largely as the only symptom on certain trees. It may be a phase of leaf spot or a separate disorder. Early and severe defoliation may result in a heavy fruit drop or in a yield of poor-quality or sunburned fruit.

Trees grown from stock which showed symptoms in the nursery may be dwarfed, and usually they are not satisfactory, since terminal growth is decidedly short. On less severely affected trees growth may be nearly normal. No particular effect on bloom or fruit set is noted except in the most severe cases. On some affected trees there is distinct evidence that varying amounts of chlorosis accompany leaf spot. When this occurs, tree vigor is further reduced and usually a tendency toward a rosette condition is apparent. Recent observations indicate that expression of Italian Prune leaf spot is less severe on trees on myrobalan rootstock than on those on peach rootstock.

Diagnostic or Unusual Characteristics

Perhaps the most striking characteristic of Italian Prune leaf spot is the severe form, which is erratic in occurrence. Some trees are affected so severely that they are dwarfed and their yield is greatly reduced. Other trees are less severely affected, but the yield and the quality of the fruit are impaired even when tree growth is only slightly affected. The severity of effect on individual trees varies widely from year to year.

Transmission

The disorder has not been transmitted, but it can be easily bud-perpetuated (*16*).

Control Measures

Propagating wood should be taken from healthy stock only. Removal of affected trees is recommended only when the orchard is young or after the trees develop severe symptoms and it has been shown that contributing factors cannot be controlled.

Remarks

Leaf spot is regarded as probably a genetic abnormality even though it is influenced greatly by soil and weather conditions. Many observations indicate that the disease may be more severe on trees near barnyards, chicken runs, or driveways and during very hot, dry seasons. It is believed that a rather complex situation exists in regard to this leaf spot and that a great deal more work is necessary to answer pertinent questions. Recent observations indicate that expression of Italian Prune leaf spot is less severe on trees on myrobalan rootstock than on those on peach rootstock.

ITALIAN PRUNE SPARSE LEAF

By Earle C. Blodgett

Name of the Disease

"Sparse leaf" is the only name used. The condition might appropriately have been called sparse fruit or unfruitfulness because affected trees bear sparsely.

Geographic Distribution

Several affected trees have been found in one orchard in southwestern Idaho. These are old trees, which, according to the grower, have shown symptoms since the orchard was started. They have been under observation since 1942 (*27*).

Economic Importance

Losses from sparse leaf are minor.

Suscept Range

The disorder has been observed only on Italian Prune (*Prunus domestica*).

Symptoms

Trees affected with sparse leaf are large and round-headed and tend to be open in type of growth. When the trees are young, growth is probably nearly normal in amount. In the orchard, affected trees can be identified easily in the leaf stage by their lack of foliage and in the dormant stage by their shape. There may be an abnormal delay of about 10 days in time of blooming (*27*) and of leafing out (*22; 25, pp. 403–404*).

Affected trees bear very sparingly. The prunes are perhaps larger than normal, but they seem to be normal in color, shape, and texture.

The foliage on affected trees is extremely sparse in comparison with that on normal trees. The sparseness seems to be due principally to the lack of small-twig growth on which leaf and fruit buds are normally borne. Apparently the lower and inner small branches either do not form or die soon after they are formed. It is estimated that the leaf surface of affected trees is little more than 50 percent of that on normal trees. The foliage is confined largely to twig terminals on the periphery of the tree. The early leaves in particular are deformed, looking somewhat similar to sweet cherry leaves affected with crinkle leaf. They are smaller than normal and have irregular margins and diffuse mottling.

Diagnostic or Unusual Characteristics

The open center of mature trees, the late blooming and foliation, and the sparseness of foliage are characteristic.

Transmission

Tests started in Idaho gave evidence of bud perpetuation, but the experimental trees were lost before it was possible to determine whether the disorder was transmissible. There is no good evidence of orchard spread.

Control Measures

Because affected trees bear sparingly, they should be replaced with good nursery stock. Budwood should be obtained from healthy trees.

Remarks

Sparse leaf is regarded at present as probably a genetic abnormality.

PEACH VARIEGATION

By Earle C. Blodgett

Other Common Names of the Disease

In the earliest report peach variegation was called calico (*15*), but after transmission attempts failed it was regarded as a chimera (*24*).

Geographic Distribution

Variegation has been observed on one J. H. Hale peach tree (original) near Caldwell, Idaho, and what appear to be the same symptoms have been seen on one Elberta tree at Emmett, Idaho. Similar variegations were observed by Reeves [46] on a J. H. Hale peach growing in the Crescent Bar district near Trinidad, Wash.

Economic Importance

Variegation is regarded as of very minor importance, and interest in it is due principally to its similarity to peach virus diseases.

Suscept Range

Variegation has been observed on J. H. Hale peach (*Prunus persica*) (original), and what appeared to be the same disorder has been seen on Elberta peach.

Symptoms

On leaves affected with variegation (fig. 69) there are rather sharply

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[46] Reeves, E. L. Unpublished data.

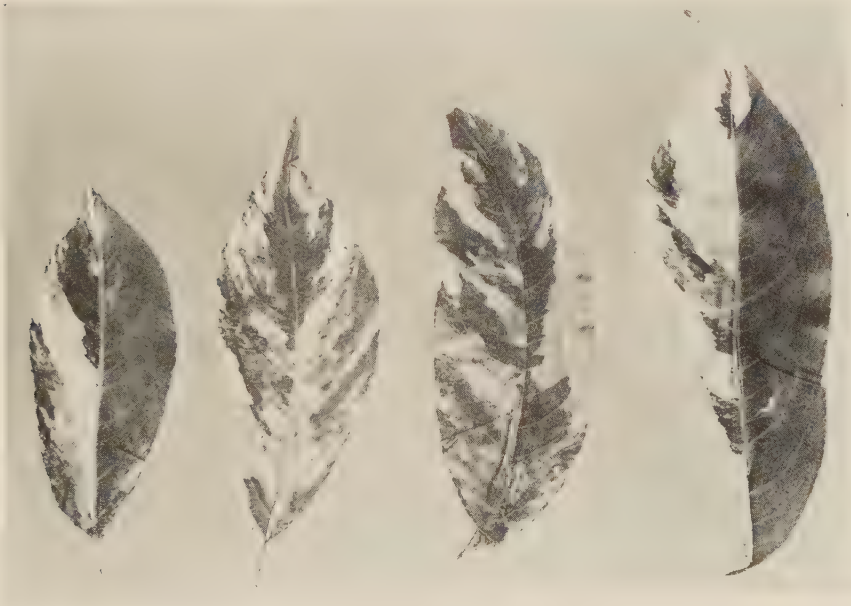


Figure 69.—**Leaves of J. H. Hale peach affected with variegation.**

delimited areas with at least three distinct shades of green. Some portions of the leaves are cream-colored to white. Often sectors or angular portions of the leaves are affected. Growth of twigs on the affected parts of a tree is materially reduced, and yellow coloration of bark tissue is sometimes associated with the condition.

Diagnostic or Unusual Characteristics

The presence of sharply defined areas of three shades of green in the affected leaf tissue is characteristic. Variegation should be distinguished from peach calico, a virus disease, in which leaf tissue is either green or white with a gradient between the two.

Transmission

Variegation is not transmissible, but it is bud-perpetuated. Three Slappey peach trees and five peach seedlings were budded with buds from trees affected with variegation, but no transmission resulted. In amount and pattern the variegation in the growth from inserted buds corresponds to the variegation in the leaves which subtend such buds.

Control Measures

Although variegation is not important at present, it is suggested that care be taken to use only wood from healthy trees for propagation.

Remarks

The exact nature of variegation is not known. It has been suggested that it is a chimera or genetic abnormality (*24*). The occurrence is apparently rare.

DEFICIENCY AND EXCESS TROUBLES THAT RESEMBLE VIRUS DISEASES

SIMILARITY OF SOME NUTRITIONAL DISORDERS AND VIRUS DISEASES

By F. P. Cullinan

Trees of the various species of *Prunus* when grown in soils deficient in one or more of the essential mineral nutrients develop symptoms frequently mistaken for those caused by viruses. Various deficiency and excess disorders of stone fruits are described and recognizable differences between them and certain confusing virus diseases are pointed out in order to help nurserymen and others working with stone fruits to make correct diagnoses of disorders found in the field.

If plants of *Prunus* species are grown in sand or water cultures from which essential elements are withheld, normal metabolism is affected and abnormal leaf and twig growth result (*322*). Some of the deficiency symptoms are specific and serve as a basis for diagnosis of like symptoms on trees grown under field conditions. In some cases the leaf pattern, for example, may be characteristic of a particular element deficiency, but in other cases it alone is not diagnostic. Thus, when iron, magnesium, manganese, or potassium is deficient varying degrees of reduction of leaf chlorophyll are apparent (fig. 70). Additional symptoms are needed along with the particular type of chlorosis to differentiate these deficiencies. Virus diseases are also responsible for varying degrees of leaf chlorosis. The pattern of the chlorosis or the markings produced in the leaf are diagnostic of some viruses that produce characteristic so-called ring spots. The basic cause of chlorosis may be nutritional, whether chlorosis is due to the lack of a mineral element or to the presence of a virus or a virus complex, because both disturb the normal metabolism of the plant. Similarly, other symptoms of mineral deficiencies and virus diseases may be due to functional disturbances.

Peach trees affected with the virus disease called phony have leaves that are darker green than normal for a variety. The leaf blade of phony-affected Elberta is more flattened than normal, and it is not wavy or fluted like the lamina of a normal leaf. Similar symptoms are observed on peach trees grown in a medium deficient in phosphorus. It has been shown, however, by chemical analysis that peach leaves from phony trees are not deficient in phosphorus.

A marked symptom of zinc deficiency in the soil is the rosette and little-leaf condition. This symptom is diagnostic of zinc deficiency on all stone fruits. Lack of other essential elements in the soil does not produce similar growth effects. Trees affected with the virus disease called "peach rosette," as the name implies, also have little leaves in rosettes (pl. 2, *A*). Other symptoms of zinc deficiency help to distinguish it from peach rosette.

"Dieback" is a general term referring to death of the growing points or the ends of upright branches. Sometimes the affected part may be 2 or 3 feet

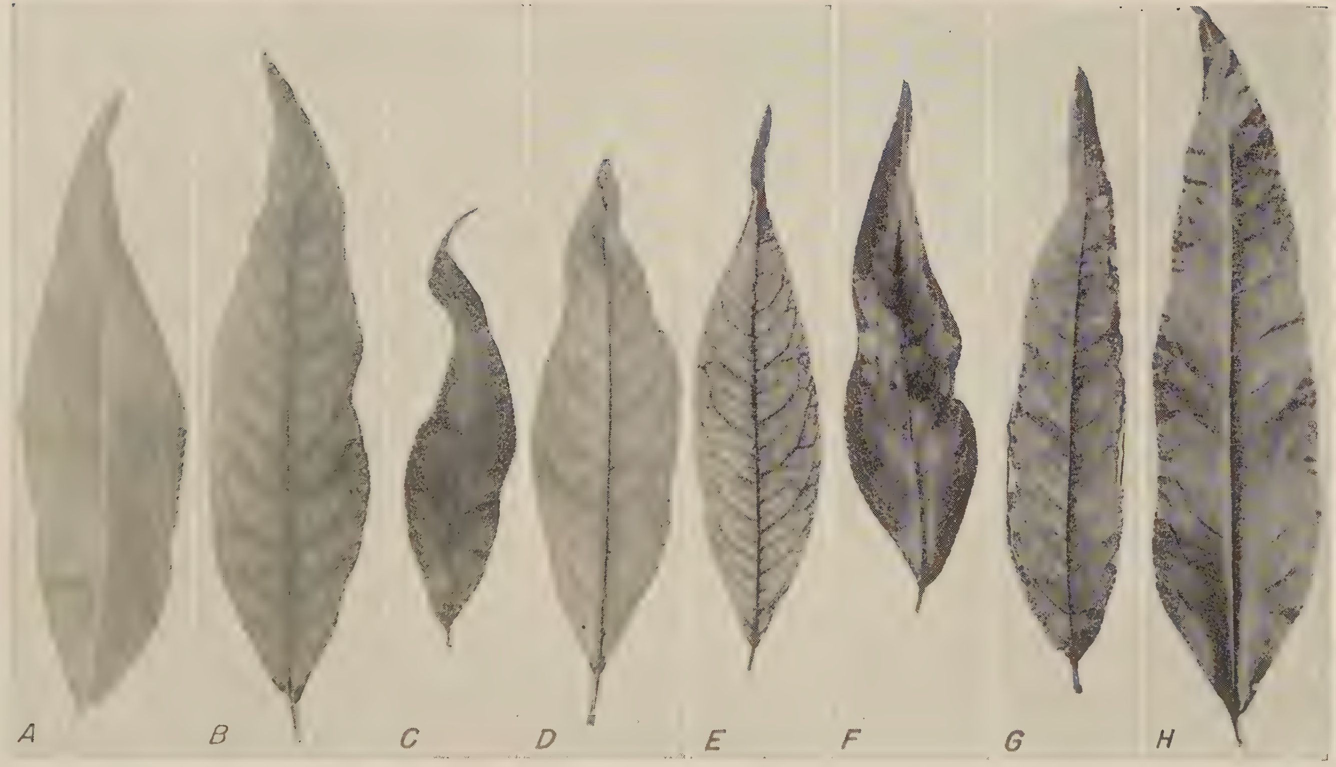


Figure 70.—Elberta peach leaves showing symptoms of mineral deficiency when trees were grown in sand cultures with indicated element withheld: *A*, Phosphorus; *B*, manganese; *C*, nitrogen; *D*, iron; *E*, magnesium; *F*, calcium; *G*, potassium. *H*, Comparable leaf from tree grown on a complete nutrient.

long. Dieback may be caused by mineral deficiencies or by virus and fungus diseases. In cases of severe zinc deficiency the little-leaf or rosette condition may be observed when the leaves first open and shedding of leaves and dieback (see fig. 76) may occur later in the same season or in the following one. Thus, another symptom characteristic of zinc deficiency is dieback. Similarly, when boron, calcium, or copper is deficient, death of the growing point may result. New growing points arise back of the injured area and clusters, or rosettes, of shoots result. These in turn may die back. In California prune trees grown in potassium-deficient soil frequently show dieback.

One of the characteristics of leaves on trees suffering from potassium deficiency is puckering or crinkling of the tissue of the blades near the midrib due to the unequal growth of cells (see fig. 71, *B*). Leaves of trees affected with the peach mottle (pl. 12, *D*) show a similar characteristic. Another symptom of nutritional disturbance noted particularly on the leaves of peach trees showing potassium deficiency is the tendency of the leaves to curl upward and roll in from the edge (see fig. 72, *B*). Rolling is observed also on trees affected with such virus diseases as western X-disease, little peach, and peach yellows. It is therefore apparent that rolling of the leaf alone is not a diagnostic character. Leaves of peach trees that have their root systems disturbed by mechanical injuries, a high water table, root or stem borers, or other causes have a tendency to roll. In diagnosing troubles suspected of being due to a nutrient deficiency, it is therefore necessary first to eliminate the possibility of factors other than nutritional ones as contributing causes. When this is done, such symptoms, for example, as leaf rolling, chlorosis, marginal scorch, crinkling, and spotting that are characteristic of potassium deficiency, become reliably indicative of the cause of the disorder.

Stone fruits, particularly peach, plum, and cherry, show shot holes in the leaves as a result of injury. This condition may be brought about by a mineral deficiency, a virus, a fungus, a bacterial infection, or a toxic chemical, but it is characteristic of some varieties. In general, when leaves of stone fruits show localized spotting from any cause, the injured or necrotic tissues drop out, leaving holes. This has been mentioned by some observers in the case of severe nitrogen and potassium deficiencies. Similarly, leaf spot fungi or bacteria may cause spots. Furthermore, materials present in toxic amounts, either in the soil or on the leaf as a result of spraying, may cause definite injury and produce shot holes. Accumulation of arsenic in the soil in toxic amounts, for example, causes typical shot hole and other leaf symptoms and marked defoliation, but the fruits hang on the tree (see pl. 27, *E*, *F*.). Soluble arsenic on the leaf resulting from the use of insecticides in spraying may result in the same kind of leaf symptoms and in defoliation if the injury is severe. In the case of the western X-disease shot hole is also one of the symptoms and marked defoliation may occur. With this virus disease, however, fruits also drop; this effect is in contrast with that of arsenic toxicity. Late in the season a few varieties of peach may develop in the leaves shot holes that apparently are not caused by nutritional or disease disturbance. An example is the Iron Mountain variety. Under some conditions this variety develops light-gray leaf spots which fall out, giving a leaf condition similar to that on trees affected with western X-disease. The leaves, however, do not fall prematurely.

In the Western States, where both zinc-deficiency disorder and western X-disease occur on stone fruits, it is sometimes difficult to determine the cause of the symptoms noted, especially when both troubles may be present on the same tree. Thorne and Wann (*315, 315a*) from observations made in Utah pointed out the following differences and similarities:

1. Western X-disease does not cause reduction in leaf size; zinc deficiency causes little leaf.
2. Western X-disease causes the leaves to curve downward and to roll in from the edges; zinc-deficiency leaves do not curve downward, but have wavy, crinkly edges.
3. With western X-disease the leaves may drop off in midseason, resulting in completely bare twigs; in severe cases of zinc deficiency, defoliation may occur but a rosette of small leaves is retained at the tip of the twig.
4. The chlorotic and red markings caused by western X-disease tend to be parallel with the leaf margin and extend across the lateral veins; in zinc deficiency the leaf markings are in general at right angles to the margin and in the areas between the lateral veins.

Thorne and Wann concluded that when all these general characteristics are taken into consideration a correct diagnosis of the disease is usually possible.

Pruning, fertilizing, and otherwise providing good cultural conditions for affected trees may correct the trouble if it is the result of a nutrient deficiency. On the other hand, symptoms of virus diseases may be accentuated by these treatments. Thus, any method of stimulating growth is a way of determining whether the disorder is nutritional or virus in nature. Leaf sprays may be very effective in indicating quickly the cause of some disorders, particularly those resulting from the lack of such elements as iron, manganese, copper, or zinc. Definite proof of the virus nature of a disorder may be obtained by transmission studies. When buds are taken from affected trees and inserted into healthy ones and the latter develop typical disease symptoms, this is conclusive proof that the disorder is infectious.

Analyses to determine the nutrient content of leaves of trees showing deficiency symptoms are helpful in diagnosis. In fact, where symptoms are not sufficiently distinct for visual diagnosis, leaf analyses may prove to be the most dependable criterion. More data have been obtained on deficiency disorders of peach than of other stone fruits. A mild case of potassium deficiency of peach trees growing on a sandy loam where the only symptoms were light-green leaves of small size may be used as an example. The potassium content of the leaves was found to be 1.25 percent on a dry-weight basis early in the season. When potash was added to the soil and the leaves were sampled late in the growing season, the potassium content had increased to 2.5 percent. The leaves were then bright green and the new ones were normal in size. Peach leaves with marked symptoms of potassium deficiency usually show a potassium content of about 1 percent or usually below on a dry-weight basis (*284*). Similarly, with other nutrient deficiencies, such as nitrogen, phosphorus, and manganese, leaf analyses are a valuable aid in diagnosis.

Excess salts in the soil may cause nutrient disturbances that result in leaf symptoms somewhat similar to those caused by nutrient deficiencies or by virus infections. Injury to stone fruits has been observed in California and other Western States in orchards irrigated with saline water. Fluctuations in the water table may be responsible for a movement of salts accumulated at lower levels in the soil to the root-zone area of a tree. Leaf scorch of stone fruits caused by injury from excess sodium (see pl. 27, *C, D*) is an example of the kind of disorder produced.

It is apparent that it may not always be easy to make a prompt, correct visual diagnosis of fruit-tree disorders in the field. In some instances the symptoms of a particular deficiency or a virus disease may be clear-cut and easily recognizable. In other cases, where the disorders are mild and the symptoms are confusing, it may be necessary to study the cause.

There are various methods available for determining the nutrient status of trees. Considerable data have been accumulated to show the value of using the leaf as an index of nutrient availability. Soil analyses may not be reliable since they may show an ample supply of some elements in the soil solution, but for various reasons these nutrient elements may not be available to the plant. In some cases a qualitative analysis may be sufficient to indicate the probable deficiency. There are various well-known quick-test methods that may be employed to confirm suspected deficiencies (*285*). A quantitative method, however, is usually more dependable. Chemical, colorimetric, spectrophotometric, and spectrographic analyses have been worked out for all the essential mineral elements.

The detailed descriptions of the nutrient disorders (pp. 222 to 251) and of virus and viruslike diseases (pp. 1 to 214) give more complete information that should help in the identification of abnormal growth conditions. The key (p. 220), which points out differences in the nature of nutrient disorders and their appearance on young and older leaves, should be helpful in differentiating disorders with one or more symptoms in common on peach. A valuable key has been published by Wallace (*319*) as a guide to the diagnosis of mineral deficiencies in various plants.

Some nutrient deficiencies now occur in stone-fruit-producing districts where they were not observed in the past. Certain growth abnormalities are noted in some orchards and not in others growing relatively close by on the same soil. It is possible that additional districts may be involved in the future. Soil-management, fertilizer, and irrigation practices, together with heavy production, may result in the occurrence of deficiency disorders, particularly on soils poorly supplied with essential elements.

KEY TO NUTRIENT DEFICIENCIES IN PEACH TREES

By O. W. Davidson

I. Symptoms in the early stages of the deficiency general on the whole tree or tending to be localized on the older leaves of current-season growth.

A. Symptoms rather generally distributed, but most prominent on lower leaves of current-season growth; without areas of dead tissue on leaves except when deficiency is advanced and severe.

1. Leaves yellowish green; discoloration beginning on old, mature leaves and progressing toward tip. Reddish and purplish-red discolored leaf spots. After prolonged deficiency, twigs hard and slender and leaves small .... **Nitrogen**

2. Young and nearly mature leaves dark green; mature leaves tan or ocher to dark green. Old leaves usually mottled, with light-green areas between dark-green veins. Progressive defoliation of mottled leaves from bases toward tips of twigs. Abnormal amounts of purplish pigment in stems and leaf petioles, especially during cool summer weather. New twigs slender and leaves small and strap-shaped if deficiency continues .... **Phosphorus**

B. Symptoms appearing first on mature or lower portion of current-season growth and occurring as mottling or chlorosis; with or without spots, blotches, marginal scorching, or other dead areas on leaves.

1. Dead areas on leaves, varying in size from very small spots or dots to patches or extensive marginal scorching. Leaves usually crinkled and somewhat curled. Dead areas developing first on mature leaves near middle or lower half of current-season growth. Twigs usually slender. Fruit buds sparse on twigs .... **Potassium**

2. Dead areas occurring as fawn-colored (or dull-brown if exposed to rain or dews) patches on most mature, large leaves. Progressive dropping of affected leaves toward the tips of current-season twigs. Defoliation severe, leaving tufts or rosettes of thin dark-green leaves at the terminals **Magnesium**

3. Leaves at tips of new growth small, narrow, more or less crinkled, and chlorotic; twigs slender, with very short internodes at tips and therefore rosettes of leaves. Progressive defoliation from bases to tips of twigs .... **Zinc**

II. Symptoms appearing first on young tissues and tending to be localized at the terminals of twigs, which die back from terminals; severe necrosis on newly developed or nearly mature leaves.

A. Immature leaves, especially the first one or two on very young twigs, and those at terminals injured, particularly along the margins. Dark-brown dead areas along midribs of nearly mature leaves. Following severe injury to foliage at terminals, twigs dying back for an indefinite distance. New branches usually developing a short distance below injured tips. Symptoms always associated with extensive injury to roots .... **Calcium**

B. Leaves more or less chlorotic and wrinkled or otherwise deformed and sometimes abnormally thick and brittle, particularly at tips of twigs. In severe cases, dieback of twigs. Fruits sometimes showing necrotic areas and cracking excessively even in dry seasons .... **Boron**

C. Young leaves chlorotic, but veins usually green. In severe cases, young leaves, including veins, sometimes cream-colored and with dead areas usually at tips .... **Iron**

NITROGEN DEFICIENCY

By E. L. Proebsting and O. W. Davidson

Geographic Distribution and Economic Importance

Lack of an adequate supply of available nitrogen is by far the most common and most important cause of deficiency disorders in stone fruits. This deficiency occurs in every major fruit district in the country unless nitrogen is applied. Nitrogen is the nutrient element to which peach trees show the greatest response in growth (*283*). Conversely, when nitrogen is lacking, growth and production of fruit are greatly reduced. Stone fruits are usually grown on light soils and on sites with good air drainage, where the spring-frost hazard to blossoms is reduced. Peaches, however, are grown on a wide range of soils varying from sand to gravelly loam, shale, and clay. Trees on all of these soils may respond to nitrogen.

The losses sustained from lack of nitrogen are not great as measured in tree mortality. Trees survive for many seasons even though they may show severe nitrogen-deficiency symptoms. The losses from an economic standpoint in terms of growth and yield are significant, and profitable commercial production is obtained only when nitrogen is adequate (*272, 307*).

Stone-Fruit Species Affected

On a given soil in California peach (*Prunus persica*) frequently shows symptoms first, but almond (*P. amygdalus*) and Japanese plum (*P. salicina*) show them a little later. Domestica plum, or prune (*P. domestica*), may not show any marked symptoms except reduced growth (*307, 308*).

In eastern United States also peach shows the most marked symptoms of deficiency when nitrogen is lacking, but here as well as elsewhere symptoms are also shown on all other species of stone fruits where the element is not available in sufficient amounts.

Symptoms

The pale-green to yellow color of the leaves of all plants grown in a soil low in available nitrogen is a well-recognized symptom of nitrogen deficiency (*272*) (pl. 23, *A*). When the supply of nitrogen in the soil is limited, trees gradually redistribute a large portion of their nitrogen from old to new tissues. As a result, the mature and nearly mature leaves turn from dark green to yellowish green. The yellow color of these leaves gradually increases, and the petioles and veins become tinged with red. By this time, linear growth of twigs has been checked and the leaf size is reduced. Moreover, the twigs and leaves are relatively hard. In 1 to 3 weeks red or red-brown spots appear between the large veins of the leaf blades if the deficiency is acute. In advanced stages of nitrogen deficiency, many of the reddish spots develop into necrotic areas. Gradual abscission of the leaves from the base of the current-season growth toward the tip follows.

Stone fruits growing in sod usually show lack of nitrogen more quickly and severely than trees under cultivation. The small, pale-green leaves of unfertilized trees growing in sod, the early cessation of growth, and early leaf fall are characteristic symptoms of nitrogen shortage. The terminal-shoot growth is frequently short and thickened, and the fruit buds are numerous. The percentage of flowers setting fruit is greatly reduced. In case of severe nitrogen shortage the shoots also are short and of slender diameter, and the number of fruit buds is less than on normal trees. Potassium-deficient trees also frequently have small, light-green leaves similar to those on nitrogen-deficient trees. In the case of potassium deficiency, however, the terminal shoots are more slender and the internodes are long. The red spotting of leaves of severely affected trees is characteristic of deficiency of nitrogen. Lack of this essential element in the soil is the only deficiency resulting in reddish discoloration of the leaf.

Control Measures

Nitrogen deficiency is easily corrected by the application of inorganic sources of nitrogen such as ammonium sulfate, sodium nitrate, or ammonium nitrate or of organic material containing soluble nitrogen (*285, 307*) (pl. 23, *A, C*).

POTASSIUM DEFICIENCY

By Damon Boynton, O. W. Davidson, and Omund Lilleland

Geographic Distribution and Economic Importance

Potassium-deficiency symptoms have been observed on various stone fruits in all counties in the fruit belt of western New York (*275, 277*). Potassium deficiency is a commercial problem in only a small part of the stone-fruit orchards, where it may retard development of trees, reduce productivity, and make fruits inferior in size and quality. The disorder has been reported on peach also in South Carolina (*309, 310*), Maryland (*284*), Pennsylvania (*287*), and New Jersey (*285*). In fact, it appears to have been found in many orchards in the Coastal Plain and Piedmont regions extending along the Atlantic seaboard from the Carolinas to New York and the Niagara Peninsula of Canada. Symptoms of potassium deficiency have also been noted in peach orchards on light sand in Michigan and on peach in Indiana and sour cherry in Wisconsin. Disorders caused by potassium deficiency are found in more peach-producing districts than those caused by other essential elements except nitrogen. While the number of trees affected in some orchards may vary from a few to many, the acreage involved in the peach sections of eastern United States is relatively small. In California disorders due to potassium deficiency have been limited to prunes in the foothill districts west of Morgan Hill and Gilroy and to a few localities near Chico (*296, 299*).

Stone-Fruit Species Affected

Peach (*Prunus persica*), sour cherry (*P. cerasus*), and domestica plum (*P. domestica*) are all affected with potassium deficiency.

Symptoms

Leaves of potassium-deficient trees usually look normal in spring and early summer except for being lighter green and having somewhat the appearance of leaves low in nitrogen. Symptoms are more prevalent and severe in dry summers than in wet ones. They are more severe on heavily loaded trees. They occur on trees growing on various kinds of soil, whether or not the subsoil is well drained. Although young trees are more commonly affected than mature ones, symptoms have been noted on bearing trees of plum, peach, and sour cherry.

Peach.—The initial symptom of insufficient potassium on peach trees is a slight yellowing of the leaves. Inasmuch as this symptom is similar to that of a lack of nitrogen (pl. 23, *A*, *B*), it is not diagnostic. The older leaves on affected trees are lighter green than those on unaffected trees, and under good cultural conditions they may start to fold or roll toward the upper surface (pl. 23, *B*). Some interveinal chlorosis may be apparent, and there may be pronounced wrinkling at the midrib (fig. 71, *B*). As the season progresses, the margins of the folded leaves fade in color and turn brown (pl. 24, *B*). Later, small straw-colored necrotic spots and blotches develop within the blades (fig. 70, *G*) and straw-colored necrotic stripes appear along the margins of leaves on the midportion of the current-season growth (pl. 24, *C*). After exposure to dews and rain the necrotic tissue becomes dull brown. The necrosis due to potassium deficiency has been commonly called leaf scorch. If the deficiency continues, necrotic areas develop on leaves progressively toward both the tips and bases of the stems. New growth continues to develop slowly at the tips, and considerable rolling is noticeable. The twigs are usually very

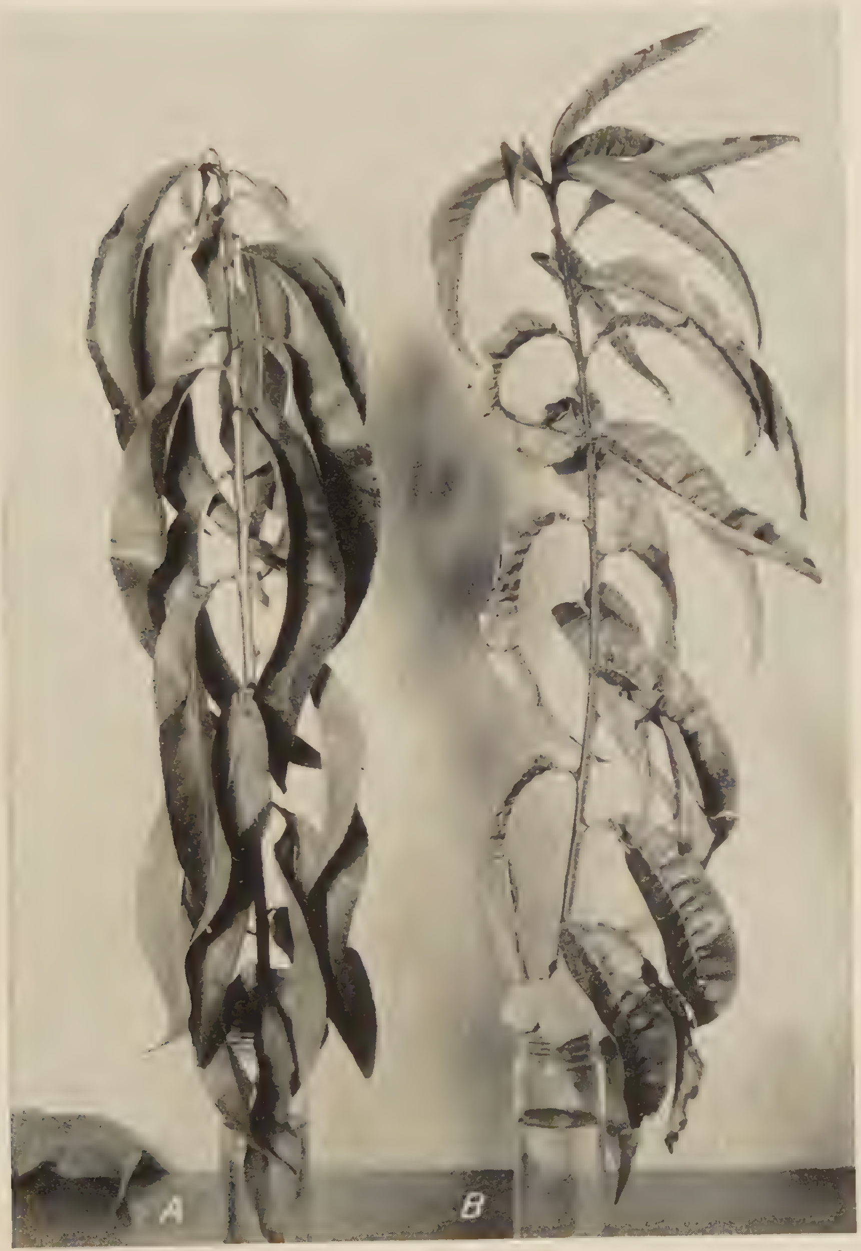


Figure 71.—Terminal shoots from Elberta peach trees in orchard at Beltsville, Md.: *A*, Adequately supplied with nitrogen and potassium; *B*, adequately supplied with nitrogen. Crinkling of the older leaves is evident in *B*.

slender (fig. 72, *B;* pl. 24, *A*), and fruit buds are sparse or lacking. On severely affected trees the leaves are apt to be small (pl. 24, *B*) and the terminal growth short. Such trees are usually stunted and produce few fruits.

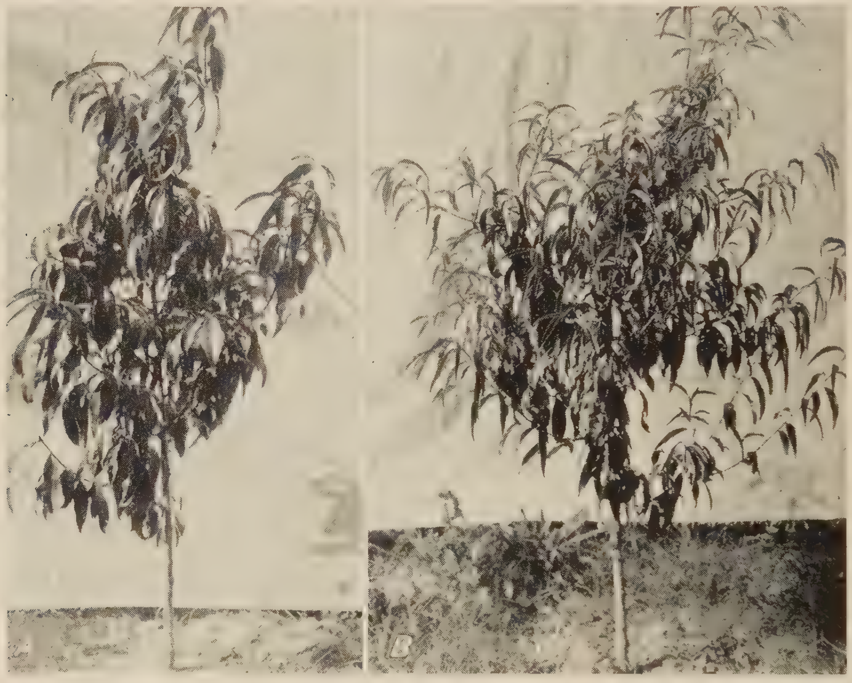


Figure 72.—Two-year-old peach trees: *A*, Adequately supplied with nitrogen and potassium; *B*, adequately supplied with nitrogen. The narrowness of the leaves and their tendency to fold are evident at the tips of the branches in *B*.

Cherry.—In July or August the older leaves of affected trees turn lighter green than those of normal trees and their margins roll upward. At the same time the leaf margin becomes discolored and the faded areas develop into necrotic, scorched lesions. Severely affected trees are apt to have small leaves and short terminal growth, to be stunted, and to produce few fruits.

Plum.—In New York the general symptoms on plum are similar to those described by Wallace (*317*). In the spring and early summer leaves of potassium-deficient trees appear normal except for being somewhat small. In July or August (depending largely on weather and degree of deficiency) the leaves on peripheral branches begin to lose their normal green color, usually fading first at the margins. As chlorosis progresses, the margins of the older leaves roll inward toward the upper surface. Finally marginal necrosis occurs. On severely affected trees almost all the leaves show a progressive development of symptoms. On moderately affected trees only the older leaves on outside branches may show the symptoms in all stages. On slightly affected trees the symptoms may develop late and on the outer branches only; they may not develop beyond the leaf roll stage. Cumulative effects of severe scorch are decreased terminal growth, delayed fruiting of young trees, and marked dwarfing (pl. 24, *E*).

In California dieback of branches, as well as leaf chlorosis (pl. 24, *D*), is a symptom. Heavy bearing usually makes the symptoms more conspicuous, and some plum trees may not show symptoms until they come into bearing. Plum trees on peach rootstocks are injured more than those on myrobalan plum (*P. cerasifera*) rootstocks. Leaves of trees that show symptoms contain less than 1 percent of potassium (dry-weight basis).

Control Measures

Prune, peach, and cherry trees with potassium-deficiency symptoms responded to applications of potassium fertilizers when they were growing on soils ranging from sandy to silty clay loams with or without well-drained subsoils (*275*). Applications of potassium chloride (muriate of potash; commercial 50 to 60 percent) at rates of 1 to 5 pounds per tree, depending upon its age, resulted in satisfactory recovery after 1 to 3 years. Fresh hay or straw mulch and manure also effectively reduced or eliminated potassium-deficiency scorch.

In California improvement was obtained with heavy applications of potash both when fruit was thinned early and when it was not thinned. Usually low-potassium areas in orchards are replanted to more tolerant crops. According to analyses of the soil by the Neubauer (rye-seedling) method, such soil areas contain less than 100 parts per million of potassium in the first 4 inches of soil (*299*).

In orchards planted on sand, clay, or gravelly loam in the Coastal Plains, peach trees responded to application of potash fertilizer (*284, 310*). Excellent results were obtained in a single season in Maryland from an application of 3 pounds of potassium nitrate containing 43 percent potash on 7-year-old trees. This fertilizer served as a source of nitrogen and potash. Equally good growth and recovery from potassium deficiency of young trees were obtained when 1 to 2 pounds of potassium chloride to the tree was broadcast on the surface of the soil.

PHOSPHORUS DEFICIENCY

By F. P. Cullinan and O. W. Davidson

Geographic Distribution and Economic Importance

Disorders of stone fruits that might be caused by lack of available phosphorus have not been as numerous as those caused by lack of some other nutrient and trace elements. It is known that phosphorus is relatively low in some soils in the stone-fruit-producing districts of the country. On these soils annual crops frequently show marked symptoms of phosphorus deficiency, and their production is limited if phosphate fertilizers are not added. Certain soils in California have a low content of available phosphorus and a high capacity for the fixation of this element. Eighteen different annual crops tested on one of the soils, an Aiken clay loam, in California failed to make satisfactory growth without the addition of phosphate fertilizer. When phosphate was added, however, there was no improvement in the growth and the yield and quality of fruits of almond, cherry, peach, prune, and other fruit trees established on this soil. Both treated and untreated trees were comparable with trees growing on more fertile soils in California (*301*).

Improvement in growth and yield of peach trees planted on a coarse phase of Norfolk sand typical of much of the sand-hill section of the Southeast was reported in South Carolina. When no phosphorus was added, the trees showed symptoms of phosphorus deficiency, but the deficiency symptoms were not as marked as those on trees on the same soil that had received phosphorus and nitrogen but no potassium (*312*). Typical potassium-deficiency symptoms were evident on them. While the omission of phosphorus in these experiments did not result in marked deficiency symptoms, lack of phosphorus was reflected in reduced growth and yield.

Similarly, peach trees on sandy soils in New Jersey have shown phosphorus-deficiency symptoms. Lack of adequate phosphorus was reported to be responsible for considerable losses to peach growers each year (*285*).

Stone-Fruit Species Affected

Phosphorus deficiency has been reported and described largely for peach (*Prunus persica*).

Symptoms

When peach trees are grown in sand culture, the first symptom is the development of a dark-dull-green to purplish-green color of the leaf. This abnormal color contrasts with the bright green of trees adequately supplied with nitrogen. The leaves show no puckering or crinkling along the midrib, and the blade is narrower than normal for the variety, leathery, and flat (fig. 70, *A*). When the phosphorus shortage is severe, the veins on the under side of the leaf and the leaf petiole may have a purplish color.

In New Jersey a compound deficiency of phosphorus and potassium has been observed to be second only to a deficiency of nitrogen in frequency of occurrence (*285*). This deficiency often occurs in young peach plantings on acid coastal plain soils. The symptoms exhibited by the affected trees range from those of a mild deficiency of one of these nutrients and a severe deficiency of the other to those of severe deficiencies of both. The trees develop a hard type of growth with relatively slender and abnormally pigmented leaves. In many instances they resemble nitrogen-deficient trees; in fact, they usually can be stimulated to make an improvement in growth for 2 or 3 weeks by heavy application of sodium nitrate or ammonium sulfate. It is possible, however, that this response is due primarily to the liberation of some potassium from the soil complex by the added cations.

Leaves on the older portions of the current-season growth droop, unlike those on normal peach trees. Young, bearing trees affected with this compound deficiency, if they set at all, produce small, dull-red fruits that are abnormally astringent. These characteristics, together with the small, rolled foliage formed on the trees, suggested many years ago the term "false little peach" for this condition.

Control Measures

Where phosphorus-deficiency symptoms have been observed under field conditions, the trouble has been corrected by application of phosphate fertilizers. Good soil-management practices, including the use of cover crops fertilized to produce a good tonnage of green manure when turned under, aid in building up the available phosphorus content of the soil.

IRON CHLOROSIS

By J. P. Bennett

Geographic Distribution and Economic Importance

Iron chlorosis (lime-induced chlorosis) is widely distributed over the western half of the United States (*274, 292*). It is of economic importance in every State lying partly or wholly west of the 98th meridian. The areas involved receive relatively low annual rainfall as compared with the eastern half of the country, where the soils are usually acid and chlorosis is rare. So far as known chlorosis in the field is limited to calcareous soils, in which iron, although abundantly present, is relatively unavailable because of their high pH. Increasing the water content of such soils by irrigation or spring rains aggravates the tendency toward chlorosis probably by raising the soil pH.

Cause and Order of Susceptibility of Stone Fruits

Plants vary widely in susceptibility to iron chlorosis induced by high soil lime. The cause of susceptibility is not definitely known, but susceptibility seems to be related to the iron requirement, extent of the root system, and the activity of the roots as indicated by respiration. Many economic plants have been practically grouped according to the amount of lime which they will tolerate without becoming chlorotic; this amount is generally lower in light than in heavy soils. The tolerated concentrations range from 1 to 5 percent for the most susceptible species on the lightest soils up to 15 to 20 percent or even higher for the less susceptible species on heavy soils.

The order of susceptibility of the stone fruits on the basis of tolerance to lime is peach (*Prunus persica*), sweet cherry (*P. avium*), plums (*P. domestica, P. salicina*), apricot (*P. armeniaca*), and almond (*P. amygdalus*) if they are on the roots of their own species. The rootstock is in general the dominant factor in susceptibility. Thus apricot or plum on peach roots are very susceptible, but on plum roots they are much less so. Trees on apricot and almond roots are also much less susceptible than those on peach roots. As compared with roots, the tops have little apparent relation to susceptibility although to a large extent they determine the iron requirements of trees. Sour cherries are affected with iron chlorosis in Utah (*315a*).

Symptoms

The principal symptom of iron chlorosis is deficiency of chlorophyll (*271, 298*) in the leaves (fig. 73). It is commonly understood that slightly to completely yellow leaves are chlorotic, but probably degrees of chlorophyll deficiency which show as lighter than normal shades of green rather than as yellowness also are indicative of iron deficiency. On leaves which are distinctly yellow the color pattern may vary from complete absence of greenness to slight yellowness between the larger veins. A common symptom is a distinct band of green along the veins, shading to yellow in the interveinal regions (pl. 25, *E*). None of the color patterns are specific for iron chlorosis; all may be found on leaves chlorotic from other causes such as potassium or magnesium deficiency.

The most distinctive characteristic of iron chlorosis is that it appears first and is most developed at the tips of growing terminal shoots. Older leaves

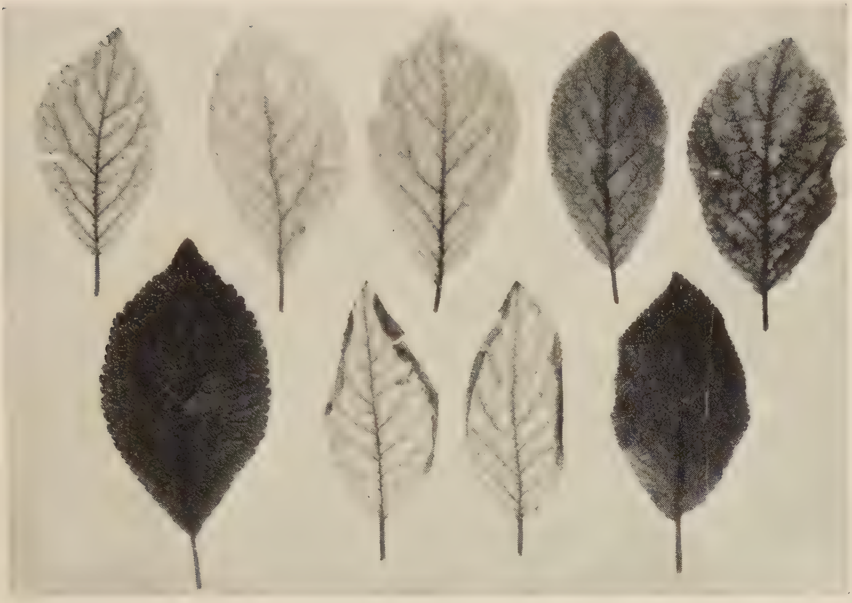


Figure 73.—**Leaves of French (Agen) prune showing the normal green and the characteristic color patterns in reduction of chlorophyll due to lack of available iron.**

are successively less chlorotic, and basal or spur leaves may be normally green. In succeeding seasons, unless remedied by treatment or more favorable soil conditions, iron chlorosis appears earlier and is more severe. Chlorosis may eventually affect not only the leaves at the base of the shoots but also those on spurs and other slow-growing branches. On trees affected with iron chlorosis the leaves are chlorotic when formed. They may subsequently become green if iron becomes available; when once fully green and fully grown, leaves do not become chlorotic as a result of iron deficiency, because the iron in leaves is highly immobile. Most of the chlorophyll is formed during the early part of the season but it generally increases further during the remainder of the season except in severely affected leaves. If iron chlorosis is light to moderate, the leaves are usually of about normal size and without lesions. But in severe cases leaf size is usually reduced and areas of dead tissue often occur along leaf margins or between the larger veins. Severe cases usually result also in shorter shoot growth and even in dying of shoots from the tips downward.

Control Measures

The control of iron chlorosis depends on making more iron available (*270, 292, 318, 320*). This can be accomplished by spraying, injection, or soil treatment. With all treatments, including spraying, applied during the leafy season, the response becomes slower as the season progresses until little or no effect is obtained from those made during the latter half of the season.

Spraying with dilute ferrous sulfate, usually of less than 1-percent concentration, is effective; but the effect lasts for only one season and spraying must be repeated during that season to include developing shoots.

Treatment by injection is done by boring holes into the live wood of the branches or the trunk or preferably in the base of the trunk or the upper part of the root below ground level, where the sapwood is thicker and healing of wounds occurs most rapidly, at intervals of 4 to 5 inches around the tree in order to get adequate distribution. A quantity of a soluble iron salt (2 to 5 gm.) is placed in each hole, which is closed by wax or a plug. Ferric citrate and ferric oxalate have been found to be especially suitable, because of the ease of handling them; but any soluble iron salt not otherwise toxic is effective.

Soil treatment is accomplished by digging trenches or boring auger holes around the tree and putting a suitable quantity of a soluble iron salt (usually ferrous sulfate) in the bottom of each trench or hole. The salt must be brought into contact with the roots to be effective; therefore holes or trenches should reach to the root zone and should be placed to give distribution around the tree. Subsequent watering aids in obtaining contact between the salt and the roots.

With both injection and trench or auger-hole treatments there is danger of killing considerable tissue, not only by the tools but by the high concentration of the salt used. Cures effected by these methods will last about 3 years if the maximum dosage that does not produce excessive injury is used. The treatments give the best results when applied during the dormant season, but they may be used when leaves are present. There is some danger of injury to the leaves, however, if large doses are used.

Permanent cure of iron chlorosis appears to require lowering the general level of the soil pH to a point at which plants are able to get sufficient iron by normal processes, whatever these may be. In highly calcareous soils this is not feasible or economically possible. Because of the aggravating effect of increased water content, better drainage and less frequent watering often improve the condition of plants. In light soils with a low lime repeated light surface applications of ferrous sulfate or sulfur or large applications of organic matter may give extended benefit. Permanent cover crops of nonsusceptible varieties appear to afford continued control whenever they can be used.

MAGNESIUM DEFICIENCY

By A. Leon Havis

Geographic Distribution and Economic Importance

Disorders in stone fruits attributable to magnesium deficiency in the soil

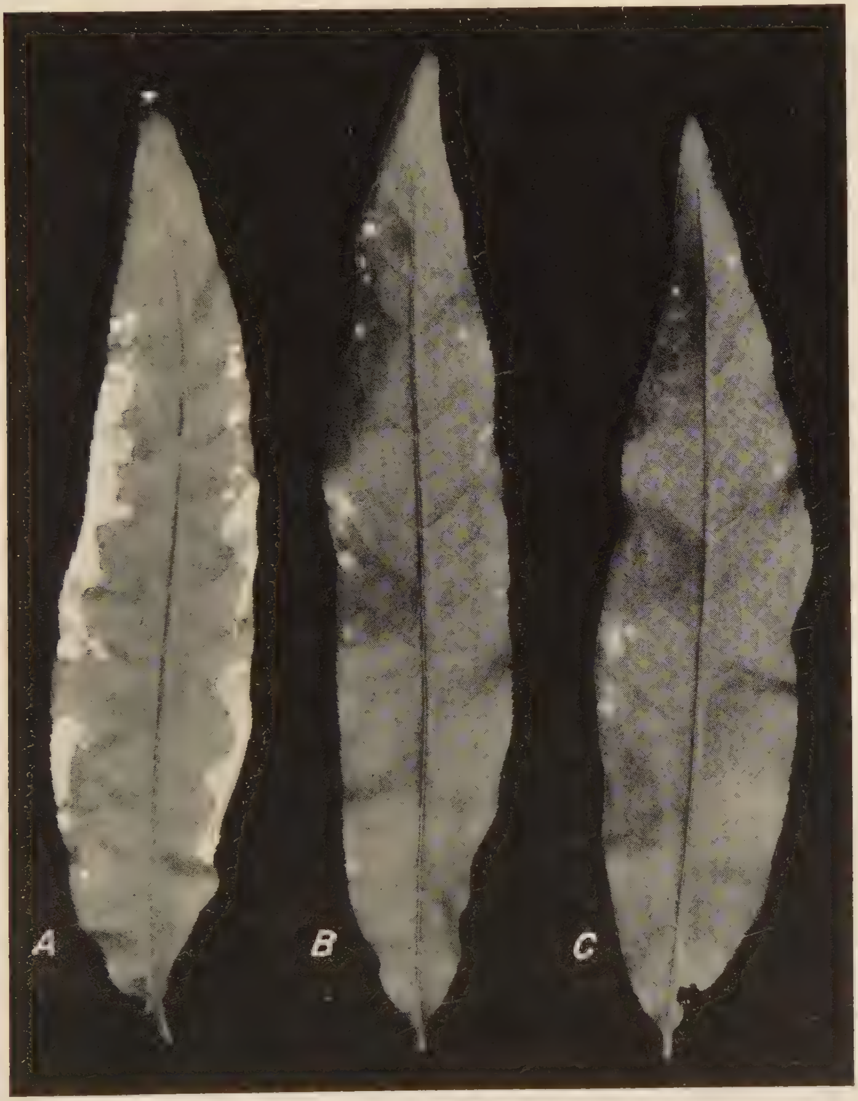


Figure 74.—Leaves from a peach tree grown in a magnesium-deficient nutrient solution: *A*, Severe symptoms; *B* and *C*, mild symptoms.

have not been reported from most of the stone-fruit districts of the country. It is known, however, that apple orchards in northeastern United States have responded to the addition of magnesium to some soils (*276*). It is not known whether apple trees are more sensitive to magnesium deficiency than stone fruits, but in an orchard on Chillum gravelly loam at Beltsville, Md., apple and pear trees (*295*) showed typical symptoms of magnesium deficiency on leaves, whereas adjacent peach trees on the same soil did not. In New Jersey magnesium deficiency is reported to occur occasionally after heavy rains in peach orchards grown on light sandy soil. In wet seasons the deficiency has been found on peach trees growing on very acid loams and on sandy loams receiving heavy applications of lime of low magnesium content (*285*).

Losses due to magnesium deficiency may result when defoliation is severe. In New Jersey winter mortality of trees occurs where peach trees lose many leaves and are weakened from the effects of magnesium deficiency (*285*). Losses due to magnesium deficiency have been insignificant in the Western States.

Stone-Fruit Species Affected

Magnesium deficiency has been noted on peach (*Prunus persica*).

Symptoms

When peach trees are grown in a magnesium-deficient nutrient solution, the older green leaves may develop light-gray- or fawn-colored spots in the area between the veins. In severe cases the spots may enlarge and take in the edges of the leaves (fig. 74). In early stages leaves may show a chlorosis somewhat like the early stages of iron chlorosis. The leaves of severely affected trees shed, from the base toward the tip, and only a few of the youngest leaves may remain near the growing tip. As the green color fades out between the veins, the leaf tissue takes on a papery gray appearance and the dead spots may enlarge and envelop the outer margin of the leaves (pl. 25, *F*). Under controlled nutrient tests in sand culture magnesium deficiency appears first where calcium or potassium is relatively high in relation to magnesium.

Control Measures

Applications of magnesium sulfate at the rate of 1 pound per tree should correct the mild cases of magnesium deficiency observed on peaches in sandy soils. Spraying the leaves with a solution of epsom salts in water may give a quicker response.

MANGANESE DEFICIENCY

By Omund Lilleland

Geographic Distribution and Economic Importance

Manganese is one of the trace elements, or micronutrients, required in relatively small amounts by all plants. Mild chlorosis of leaves caused by manganese deficiency has been observed on stone fruits in California, Utah, Michigan, and New York. A number of unpublished observations of chlorosis suspected of being caused by lack of manganese have been made in several peach-producing districts of eastern United States.

The amount of injury to trees or loss of crops has in general been negligible. In California in one orchard that was severely affected and chlorotic a crop of only 3 tons to the acre was harvested instead of a 10-ton crop that would be expected on trees of the same age with healthy foliage (*291*). Defoliation seems to be rare, and no evidence of dieback has been observed. It seems unlikely that manganese deficiency will cause any serious effects in commercial stone-fruit orchards, because it is easy to correct.

Stone-Fruit Species Affected

Manganese deficiency has been observed on almond (*Prunus amygdalus*), apricot (*P. armeniaca*), sweet cherry (*P. avium*), sour cherry (*P. cerasus*), peach (*P. persica*), domestica plum (*P. domestica*), and Japanese plum (*P. salicina*).

Symptoms

A distinct pattern has been observed on the leaves of all stone-fruit species on which manganese deficiency has been observed. The midrib and main veins with adjacent bands of tissue of varying width remain green, whereas the interveinal and peripheral areas of the leaf edges may be chlorotic (pl. 25, *A–D*). All the leaves on some severely affected trees may show some degree of chlorosis, but on other trees only a few branches may be involved. Leaf scorch may occur when the deficiency is severe. There are no marked symptoms recognizable on the fruits or twigs.

Samples of leaves of peach taken from 39 Elberta orchards and analyzed for manganese content showed a range of 293 to 6 p.p.m. (parts per million) on a dry-weight basis. Deficiency symptoms on peach generally seemed to be associated with a manganese content of less than 17 p.p.m. The changes in manganese content throughout the summer were not large, and it was concluded that samples of leaves taken at any time from June to October in California would reflect the general level of manganese in peach trees (*291*). Analyses of chlorotic leaves of 6 kinds of fruits, including peach, almond, prune, and sweet cherry, gave values ranging from 5 to 25 p.p.m. of manganese. Differences in content could not be correlated with species.

Control Measures

Manganese deficiency is easily corrected by use of leaf sprays in early summer (5 to 10 pounds of manganese sulfate in 100 gallons of water). Injection of a manganese sulfate solution into the branches and soil treatments with manganese sulfate are also effective. In a severely chlorotic orchard in California a 1-percent solution of manganese sulfate was sprayed on certain branches in the spring and in 2 weeks the sprayed branch on every tree was normal, while the rest of the tree was chlorotic. Similarly, in New York D. Boynton injected dry manganese sulfate into the branches of chlorotic sour cherry. The following spring the treated branches bore normal green leaves, whereas the untreated ones bore chlorotic leaves (pl. 25, *C, D*).

ZINC DEFICIENCY

By W. H. Chandler

Geographic Distribution and Economic Importance

In the hot interior valleys of the Pacific slope, stone-fruit trees are apt to show zinc-deficiency symptoms on sandy soils, especially if cultivation is frequent during the summer. Old sweet cherry trees are apt to show such symptoms even on loams or clay loams. At one time the losses from decline of trees and reduction in crops amounted to millions of dollars. Even in orchards that have not had zinc applications losses from zinc deficiency have been greatly decreased by the general reduction in summer cultivation; the decrease is possibly due to soil shading by weeds that are permitted to grow.

Zinc deficiency has been reported on stone fruits in Utah, Idaho, and Colorado in addition to the Pacific Coast States and British Columbia, Canada. It seems likely that it might be found on these fruits in all 11 of the Western States, since it has been reported to occur on other trees in that region. In general, zinc deficiency has occurred on sandy soils or on those treated with large amounts of organic matter. In some of the Western States disorders shown to be due to lack of available zinc were observed first on sites formerly used as corrals or other enclosures for livestock. In the Pacific Coast States the deficiencies are confined largely to the irrigated valleys of the interior. In the Mountain States deficiency of zinc has also been observed in irrigated valleys. Up to the present time zinc deficiency has not been reported on stone fruits in the eastern half of the country except in New Jersey (*282a*) and Florida. In Florida (*286*) it has been found in peach orchards growing on Norfolk fine sand. Unpublished observations indicate that zinc deficiency may occur on peach elsewhere in the East.

Susceptibility of Stone Fruits

Chandler (*280*) listed sweet cherry (*Prunus avium*) as the most susceptible of the stone fruits to zinc deficiency. Other stone fruits affected, in order of susceptibility, seem to be Japanese plum (*P. salicina*), domestica plum (*P. domestica*), peach (*P. persica*), apricot (*P. armeniaca*), and almond (*P. amygdalus*). In Utah zinc-deficiency symptoms have been observed in sour cherry (*P. cerasus*) (*315, 321*).

Symptoms

Leaf symptoms have been emphasized more than others. Irregular splashes of yellow occur between the veins, especially on leaves of the new growth in the first few weeks of spring and on long shoots during the last few weeks of summer. Most of the leaves have wider green areas along the veins and around the yellow splashes than manganese-deficient leaves have, and the yellow tends to be paler and less golden. Leaves at the end of some summer shoots may be creamy yellow, with very little green even along the veins. Leaves that develop in the first few weeks of spring at the apex of a long shoot of the preceding year may be very much smaller than normal (about 1 inch long and ¼ inch wide or less), nearly sessile, and stiff; the disease has been known as little-leaf (*281, 282, 286, 297*). The small, stiff leaves are crowded very closely into tufts (fig. *75*). Because the internodes are very short, the disease is some-

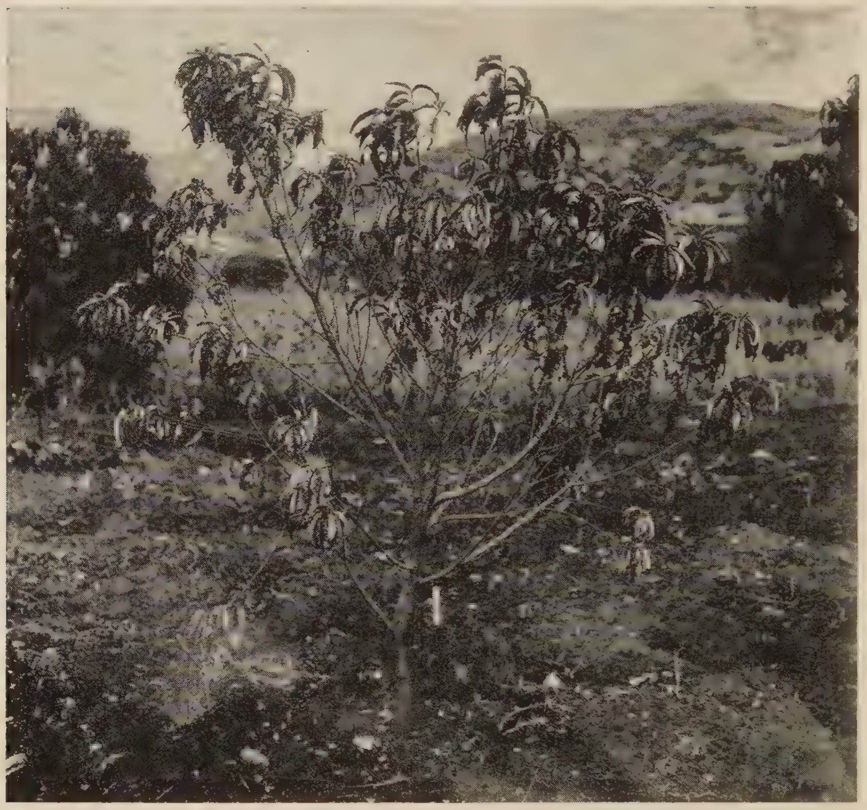


***Figure 75*.—Young peach tree showing typical rosette condition accompanying zinc deficiency, Utah.**

times called rosette (pl. 26, *A–C*). The branches may die back so much in late summer that few if any rosettes grow in the following spring, the upper shoots being water sprouts from dormant buds below the dead parts.

Such dying back of zinc-deficient cherry trees is so general that spring rosettes are rarely seen (fig. 76). Leaf mottling and very small fruits on the outer parts of branches may be the only distinctive symptoms of serious zinc deficiency.

Old almond trees may not show a distinct mottling of the leaves, and the leaves may have only a paler than normal color. Because bearing trees are pruned so little and make such short shoot growth, rosettes as well as distinct mottling are apt to be seen in early summer on water sprouts of the preceding year only. Young, vigorous, zinc-deficient almond trees, however, may show mottling and rosettes much like those on Japanese plum.

On trees of all affected species of stone fruit, the internodes of the last few inches of the longest summer shoots may be very short and the leaves may be rather small, yellowish, often much distorted, and closely packed.

In peach, apricot, plum, and cherry orchards, the most dependable zinc-deficiency symptoms occur on the fruits if the deficiency is not bad enough to prevent all fruits from setting. Fruits on the lower parts of the trees may be normal or nearly so. Those in the tops, and especially toward the ends of the branches, tend to be progressively much smaller than normal (pl. 26, *C*). In California at least, this progressive decline in size of fruits toward the ends of the upper branches seems always to be conclusive evidence of zinc

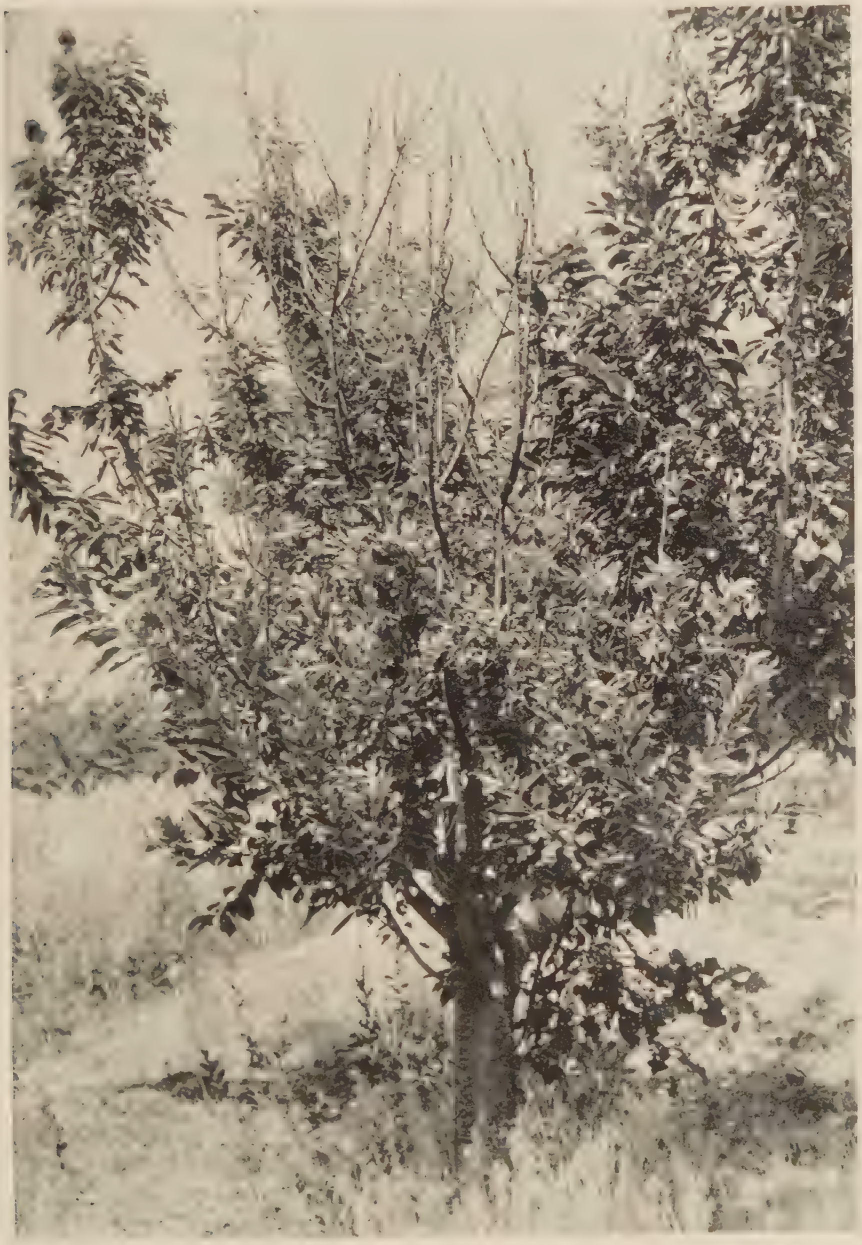


Figure 76.—Sweet cherry tree showing production of small, chlorotic leaves and severe dieback, caused by zinc deficiency, Utah.

deficiency. The fruits also tend to be abnormal in form. Those of peach and Japanese plum are more flattened than normal and constricted below the apex into a rather thick neck with a terminal beak. Apricot fruits are less flattened than normal, more nearly round, and grading downward in size to that of medium-sized cherries. Mature peaches from zinc-deficient trees break down or turn brown sooner after harvest than those from normal trees.

Control Measures

Most soils have great capacity to render zinc unavailable to trees. In some of the Pacific slope soils this fixing capacity is so great that 1,000 to 1,500 pounds of zinc sulfate applied to an acre of soil may not supply the necessary ounce or two of zinc to an acre of trees. Soil applications are therefore avoided as much as possible.

Leaf spraying with zinc-lime has not been very effective on deciduous trees. Dormant spraying with zinc sulfate alone, 10 to 50 pounds of zinc sulfate to 100 gallons of water, has been found to be the best practice that has been tried for peach, almond, and Japanese plum. The amount required depends upon the severity of the symptoms. If there is much dying back, even more than 50 pounds to 100 gallons may be advisable in dormant applications. If the zinc is applied in combination with bordeaux mixture against leaf curl or other diseases controlled by winter spraying, somewhat more zinc sulfate should be used because of the effect of the lime in the bordeaux mixture. Apricot does not respond so well as peach to dormant spraying, and therefore stronger solutions must be used. Sweet cherry trees hardly respond at all to spraying, unless the soil is one with such a low fixing power that soil treatment (500 pounds of zinc sulfate or less to the acre) is feasible.

Driving very many zinc-coated finishing nails into the trunk and branches, with the heads driven to the bark, may be the only treatment. As many as 40 to 50 zinc-coated, 1-inch-long nails for each inch of trunk circumference may be required; no nail should be nearer than a half inch to another, or the bark may be killed between them. And, of course, the nails should be rather well distributed around the trunk or branch. Great masses of gum will exude around each nail, but this seems to do no harm unless the nails are too close to each other.

COPPER DEFICIENCY

By Carl J. Hansen

Geographic Distribution and Economic Importance

Copper deficiency (exanthema) was first reported on plums in California by Smith and Thomas (*313*) in 1928. Since then additional affected plum trees have been found in a few localities in the State. Other stone fruits have not been definitely proved to be suffering from copper deficiency in California. However, in a few former corral areas, where other complications would obscure symptoms of copper deficiency if it were present, peach, cherry, and apricot have in some instances responded to applications of copper.

Copper deficiency has not been reported on stone fruits in eastern United States, but deficiency of copper in Florida soils has been shown to affect citrus (*279*).

Stone-Fruit Species Affected

Only plums (*Prunus domestica* and *P. salicina*) are positively known to be affected with copper deficiency in this country. In South Africa copper-deficiency chlorosis has been observed on apricot (*P. armeniaca*) and peach (*P. persica*) (*269*).

Symptoms

The tips of the shoots of the affected varieties of domestica and Japanese plum are killed, and the lateral buds are forced into growth. These lateral shoots may in turn die back as illustrated by the Imperial prune shoots in plate 26, *D*. The chlorosis and scorching of the Late Santa Rosa plum leaves shown in figure 77 have been identified as symptoms of copper deficiency. The trees affected were cured by applying copper sulfate to the soil. Some other varieties of plums, at least under the particular climatic and soil conditions where they were observed, showed none or practically none of the leaf symptoms. The leaves, for example, remaining on the two shoots in plate 26, *D*, are practically normal, although some scorch was observed on those that had fallen. The bark of some small branches of affected trees may be rough and corky, but the extent of such injury depends on the variety. Swellings may also occur in the regions surrounding the buds and the lateral shoots.

In many respects the copper-deficiency injury illustrated in plate 26, *D*, resembles the injury caused by excess boron. Copper deficiency, however, does not produce corky areas on the leaf petioles and veins and does not cause injury to the stems just above the buds.

Control Measures

Plum trees showing symptoms of copper deficiency have been cured by application of copper sulfate to the soil, by injecting copper sulfate into holes bored in the trunks, and by spraying the young leaves with 5–5–50 bordeaux mixture.

Soil applications have generally varied from 1 to 5 pounds of copper sulfate placed in the bottom of a circular trench or spaded in around the base of each tree, the exact quantity depending on the size of the tree and the fixing power of the soil. In a few soils with a high fixing power even considerably heavier applications were not effective.

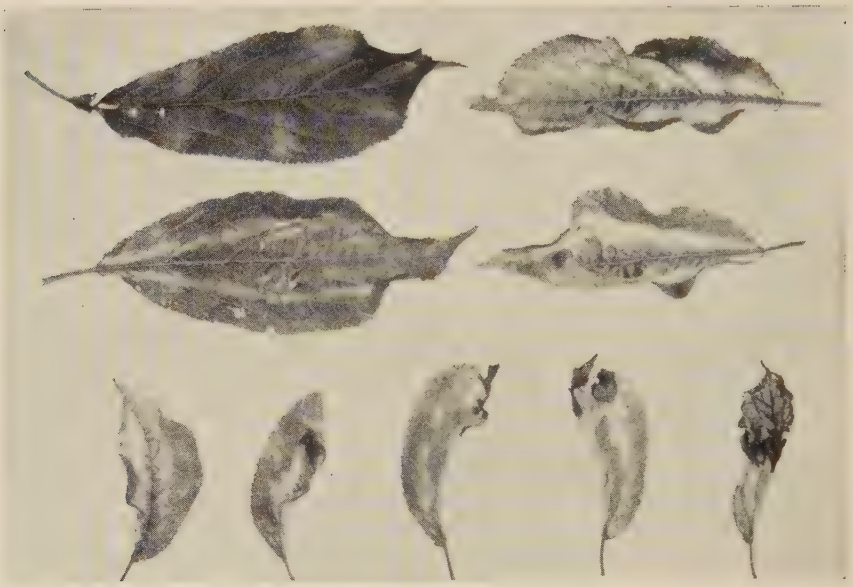


Figure 77.—**Leaves of Late Santa Rosa plum showing chlorosis and scorching characteristic of copper deficiency.**

The holes used for the injection of dry copper sulfate are spaced about 4 inches apart around the trunk, and excessive injury is avoided by keeping the material away from the cambium and the bark. This is done by inserting the copper sulfate in gelatin capsules or forcing it through a tube. Wooden plugs are generally driven into the holes.

It is likely that more copper deficiency would occur if most stone-fruit trees were not regularly sprayed with copper sprays for disease control.

CALCIUM DEFICIENCY

By A. Leon Havis

Geographic Distribution and Economic Importance

In the fruit-producing districts of this country no serious disorders in top growth of stone fruits that could be attributed to lack of calcium have been reported. In the humid region of the eastern part of the country a number of soils are acid, and satisfactory growth of many plants on these soils is obtained only when lime is added to correct the acidity and raise the pH. Many soils have been made productive, particularly for legumes, grasses, and cereal grains, when lime and phosphate were added as fertilizers. On many of the soils on which peach and other stone fruits are grown the soil is frequently acid and low in calcium. Tree fruits are known to tolerate acid soils. Calcium is an essential element required by all plants; stone fruits are not an exception. The appearance of deficiency symptoms under field conditions, however, is not marked. The addition of lime to soils low in potassium and magnesium may produce deficiency symptoms of the last two elements. The stone-fruit-producing districts of the Western States have alkaline soils, and no symptoms of calcium deficiency have been reported on stone fruits.

Stone-Fruit Species Affected

Peach (*Prunus persica*) is the only species known to suffer from calcium deficiency in orchards.

Symptoms

When peach trees are grown in a nutrient solution lacking calcium, the first symptom of the deficiency is usually decreased terminal growth; the older leaves may be of normal size, but the young leaves are usually smaller than normal. The color of the leaves is dark green, and no chlorosis is evident. Later, a large chlorotic area develops in the center of some of the younger leaves. It is a large, characteristic spot involving tissue on either side of the midrib. The older leaves later show marginal chlorosis and breakdown (pl. 27, *A*). Finally the leaves drop off at the tip; then the ends of the shoots may die back. Under controlled nutrient conditions calcium deficiency is shown in peach foliage first where potassium and magnesium are relatively high.

In the field calcium deficiency has been reported to curtail greatly root extension of peach (*285*).

BORON DEFICIENCY

By Carl J. Hansen, F. P. Cullinan, and E. L. Proebsting

Geographic Distribution and Economic Importance

Boron is one of the elements essential to all plants, but the amount required is relatively small when compared with some of the other essential elements. A trace is needed in nutrient solutions for satisfactory growth of some plants, but only a few parts per million more than needed for optimum growth may be toxic to other plants. In some of the irrigated districts of the West the concentration of boron in the irrigation water may be high enough to cause injury to stone fruits.

Boron deficiency on apricot and peach has been reported in virgin soils in British Columbia, Canada, (*291a, 304*) and in commercial orchards in the Northwest (*285*). It is known to occur on apple in northeastern United States, principally in New York and the New England States. Internal cork of apple found in orchards in the Cumberland–Shenandoah Valley (*305*) and New York (*278*) has been corrected by injection of boric acid crystals or application of boric acid to the soil. Apple orchards in southwestern Michigan also have been improved by application of borax. Even though boron is known to be deficient in soils in eastern United States, there have been no reports thus far of marked boron deficiency on stone fruits. Some breakdown in the flesh of peach fruits has been suspected of being due to lack of boron, but thus far correction of this trouble has not been effected by application of borax.

Boron deficiency has been observed on domestica plums in the Sierra Nevada foothills of California (*294*). The orchards were on soils of the Aiken and Sites series.

In orchards in the vicinity of The Dalles, Oreg., a dwarfed, chlorotic condition has been observed on the leaves of cherry, peach, prune, and apricot. The trouble was most noticeable on sweet cherry. The symptoms, which were in general characteristic of zinc deficiency, disappeared after the trees were sprayed with zinc sulfate, but better results were obtained when boric acid was added to the spray solution (*311*).

In nutrient-culture experiments with apricot in California deficiency symptoms developed when the boron concentration of the nutrient solution was 0.1 p.p.m. (parts per million). The trees showed leaf injury and dieback. When the concentration of boron was raised to 0.5 p.p.m., growth was resumed and 6 weeks later no evidence of boron deficiency was noticeable (*297*).

Stone-Fruit Species Affected

Domestica plum (*Prunus domestica*) varieties President, Giant, and Diamond are the only plums in California that up to now have shown symptoms of boron deficiency. Varieties of Japanese plum (*P. salicina*) growing adjacent to affected domestica plums appeared normal. Peach (*P. persica*) is naturally affected with boron deficiency and has been experimentally affected. Apricot (*P. armeniaca*), sweet cherry (*P. avium*), and cherry seedlings also are affected.

Symptoms

The only boron-deficiency symptoms that appear consistently on plums in California orchards are on the fruits. Brown sunken areas in the flesh may consist of a single spot or may involve practically the whole fruit. The flesh beneath the sunken parts is brown and firm, and in severe cases the browning extends to the pit. The fruits color earlier than normal fruits and drop. The number of fruits on a tree showing these symptoms may vary from a few to the entire crop. Leaves from trees showing deficiency symptoms in the fruit contained 25 p.p.m. or less of boron, whereas leaves from normal trees contained 30 p.p.m. or more. Leaf concentrations ranging from 26 to 29 p.p.m. were obtained from both normal and deficient trees.

In British Columbia the outstanding symptoms of boron deficiency on peach are failure of buds to break in the spring and dying of twigs, branches, and sometimes even entire trees (*304*). When peach is grown in a nutrient solution lacking boron the first symptoms appear at the terminals of upright shoots or at the end of the leader. The terminals die back, dark-green water-soaked spots appear in the bark, gum may exude, lateral buds develop, and the resulting new shoot growth also dies back. Small irregular spots appear in the leaf, die, and drop out. Most of the affected leaves are chlorotic. Injured leaves fall off rapidly from the tip toward the base (*322*). On the stems dark-brown, corky spots appear (*289, 322*). Cherry and peach seedlings in sand cultures made good growth when the concentration of boron in the nutrient solution was 1 p.p.m. Both of these fruits showed injury when the concentration was 5 p.p.m. (*289*). In British Columbia, however, twigs from peach trees that showed signs of boron deficiency had a boron content of 4 to 8 p.p.m. (*304*).

In the Northwest the apricot shows the following fruit symptoms (*277a*): Internal browning and corky tissue developing in the stone area; cracking, shriveling, surface browning, and constrictions.

Control Measures

Boron-deficiency symptoms on plums disappeared when borax was broadcast on the surface of the soil in late summer at the rate of 0.5 pound to the tree, or approximately 50 pounds to the acre. It is estimated that the effect of a single application may last 3 years. Some boron injury occurred when borax was used at the rate of 1 pound to the tree (*294*). Applications of 0.5 pound of borax per tree have corrected boron-deficiency symptoms on apricots.

BORON-EXCESS INJURY

By Carl J. Hansen

Geographic Distribution and Economic Importance

Injury to stone fruits by an excess of boron has occurred in approximately one-fourth of the counties of California. The districts where boron injury has been reported are scattered mostly in the western part of the State. They consist of single orchards irrigated by a well high in boron or of several orchards. Excess boron has sometimes been found occurring naturally in the soil where irrigation is not practiced (*288, 290*).

Stone-Fruit Species Affected

Almond (*Prunus amygdalus*), apricot (*P. armeniaca*), sweet cherry (*P. avium*), peach (*P. persica*), Japanese plum (*P. salicina*), and domestica plum (*P. domestica*) are known to be injured by excess boron.

Symptoms

Many kinds of plants show marginal leaf scorch when grown in soil containing excess boron, but this scorch does not occur on apricot, peach, plum, cherry, and almond. On plum, apricot, peach, and almond the principal injury is to the shoots of the current-season growth. The French (Agen) prune shoot in plate 27, *B,* shows how the shoots die back at the tip and the cracked, corky bark, a symptom of the excess. Sometimes gum may also be exuded from the injured parts. The short section of a shoot illustrates the tendency for the injury to be most severe just above the buds. Corky areas similar to those on prune petioles and on the large veins of the lower side of prune leaves (pl. 27, *B*) are also found on apricot (fig. 78). On peach and almond the leaf injury is limited to the midribs. If other conditions are normal for growth, the lateral buds on injured apricot, plum, and peach shoots start to grow, only to be killed back later. When apricot trees are dormant, the injured branches look like the shoots shown in figure 79. Enlargements like those at the nodes do not appear on peach. Cherries are injured by too much boron, but symptoms such as those just described are rarely found. Cause of injury can be determined by analysis of soil and plant parts and by observation of typical symptoms on nearby plants (*293*).

Injury sometimes occurs on fruits of peach and occasionally on those of apricot. The injured areas on the peach fruits are dark brown and woody; they vary in size, but a single one may involve over half a fruit and extend from the surface almost to the pit. The injury on apricot fruits consists of dark, circular areas approximately ¼ inch in diameter.

Control Measures

Unfortunately there is no cure for excess boron except the adequate use of satisfactory irrigation water.

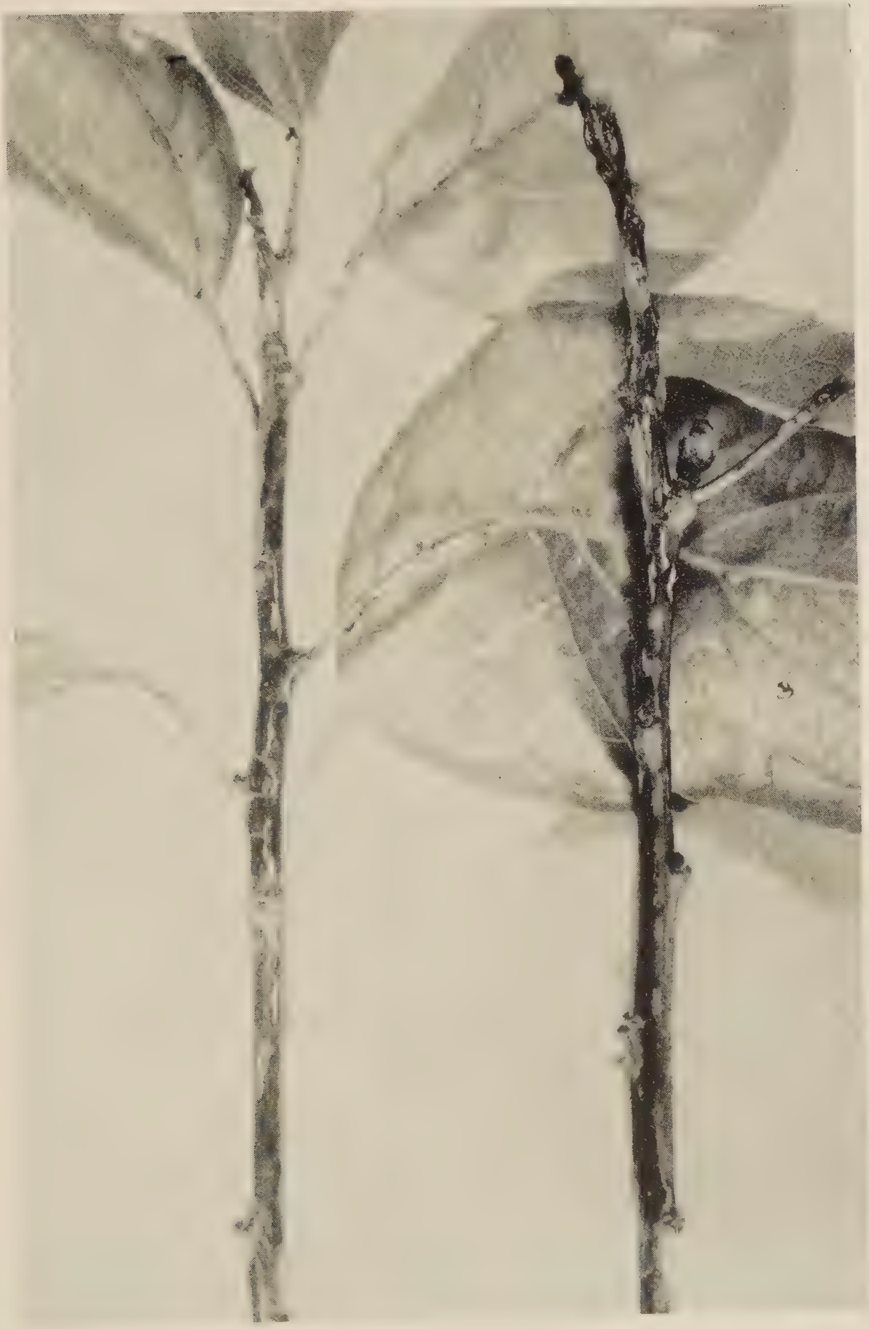


Figure 78.—Apricot shoots injured by excess boron, showing dieback at tips and cracked and corky bark.

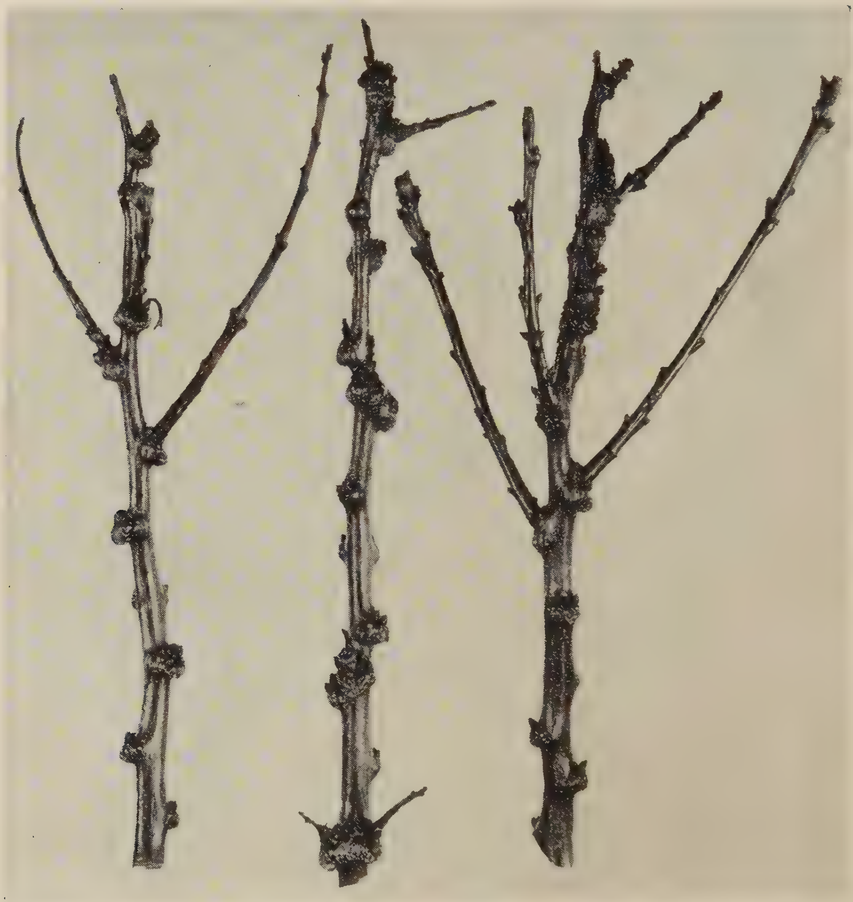
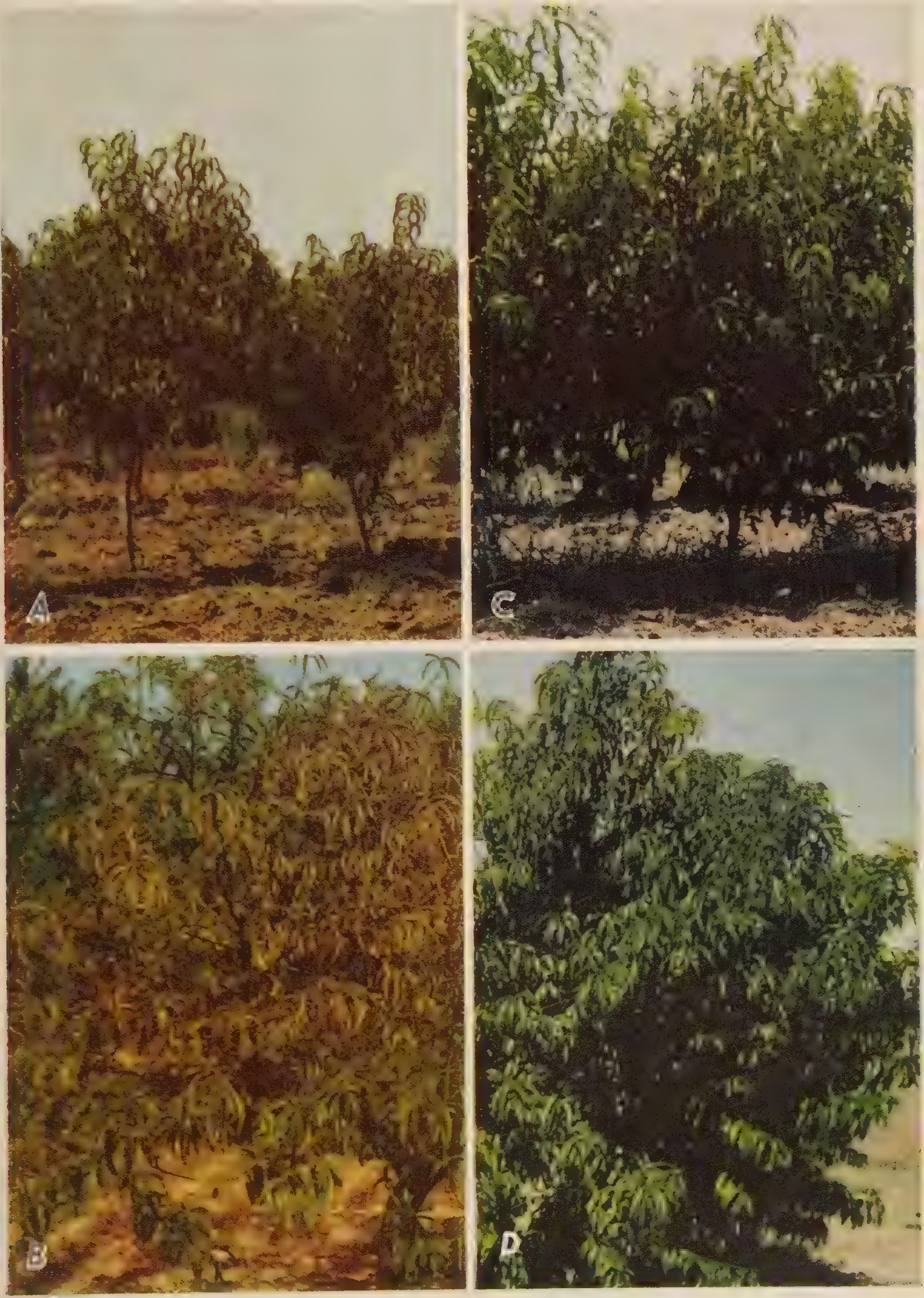
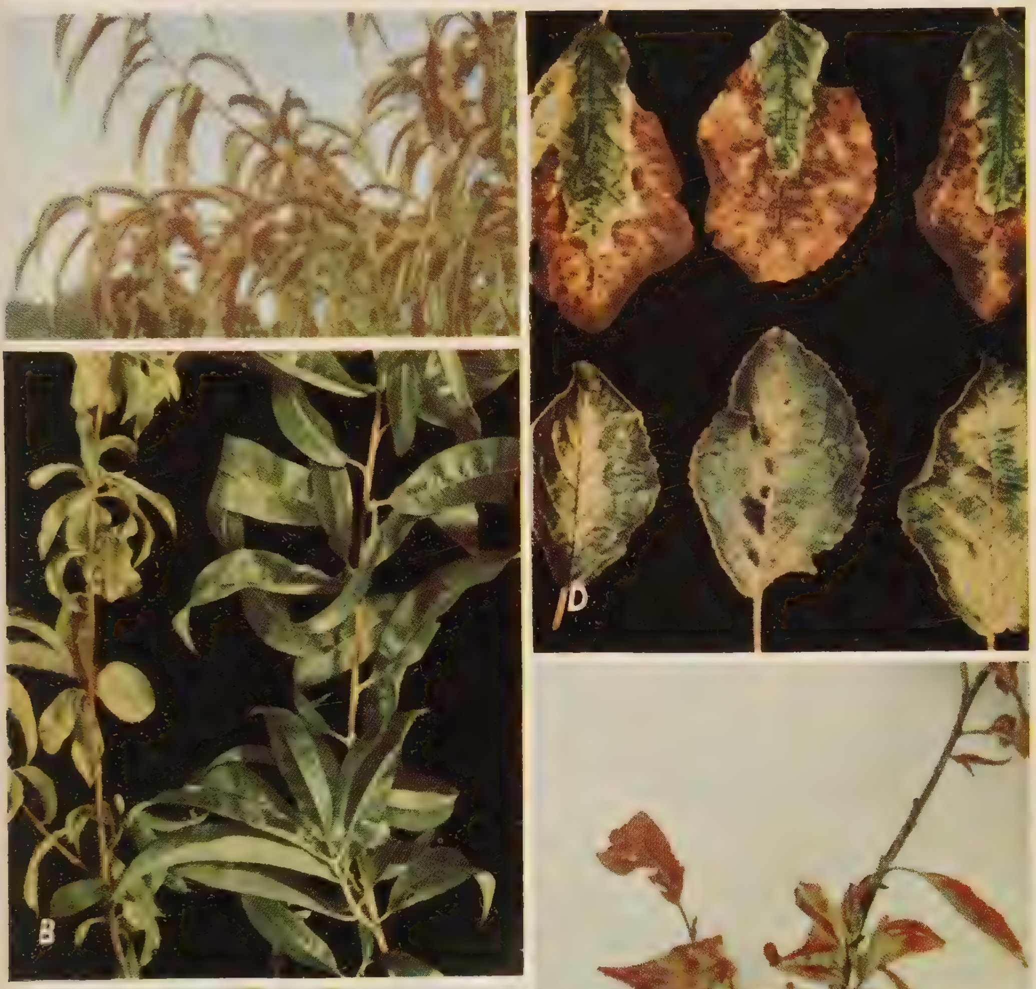


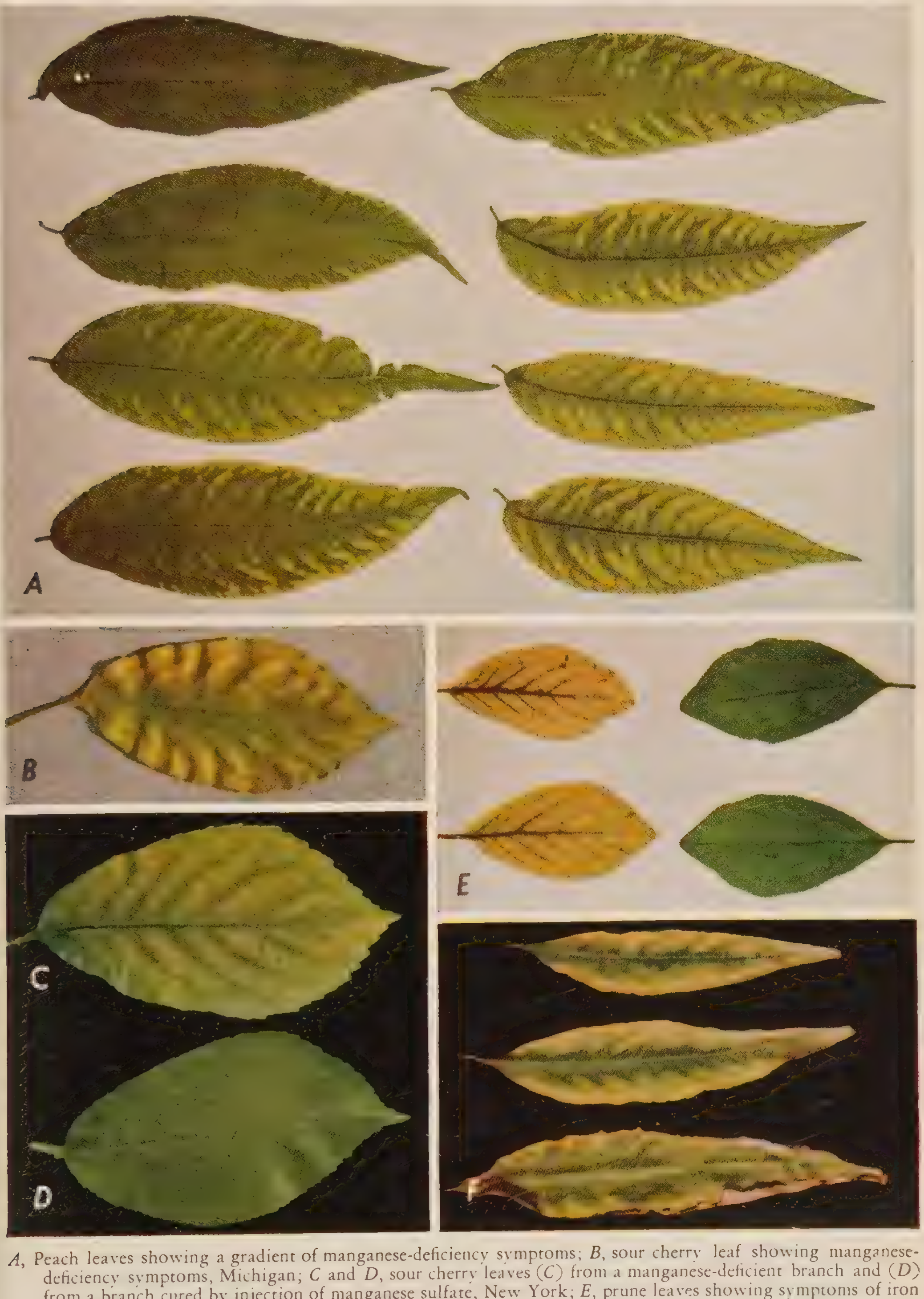
Figure 79.—Dormant apricot shoots, showing at nodes enlargement caused by excess boron.



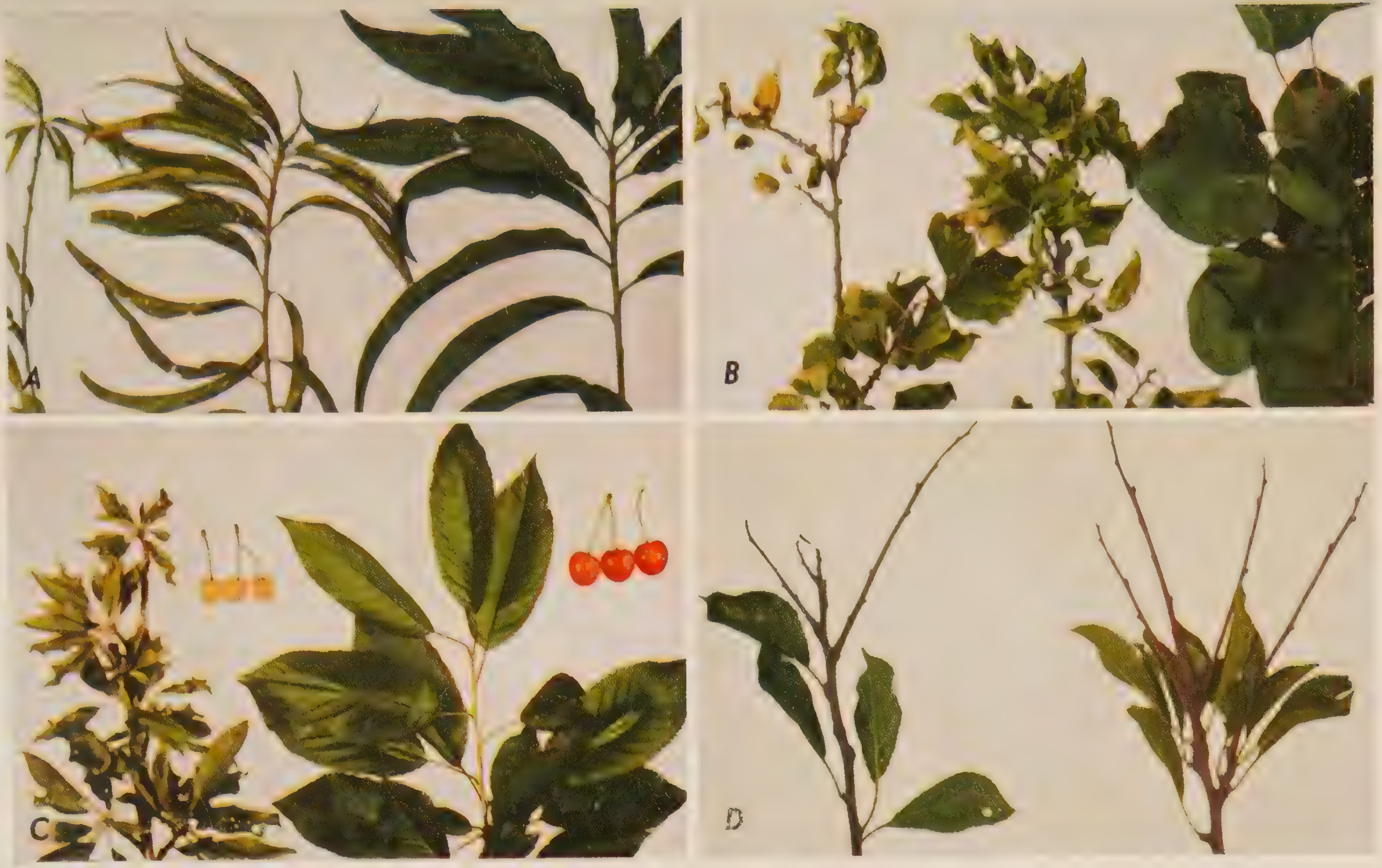
A, Peach trees showing symptoms of nitrogen deficiency; *B*, peach tree showing symptoms of potassium deficiency; *C* and *D*, comparable normal trees.



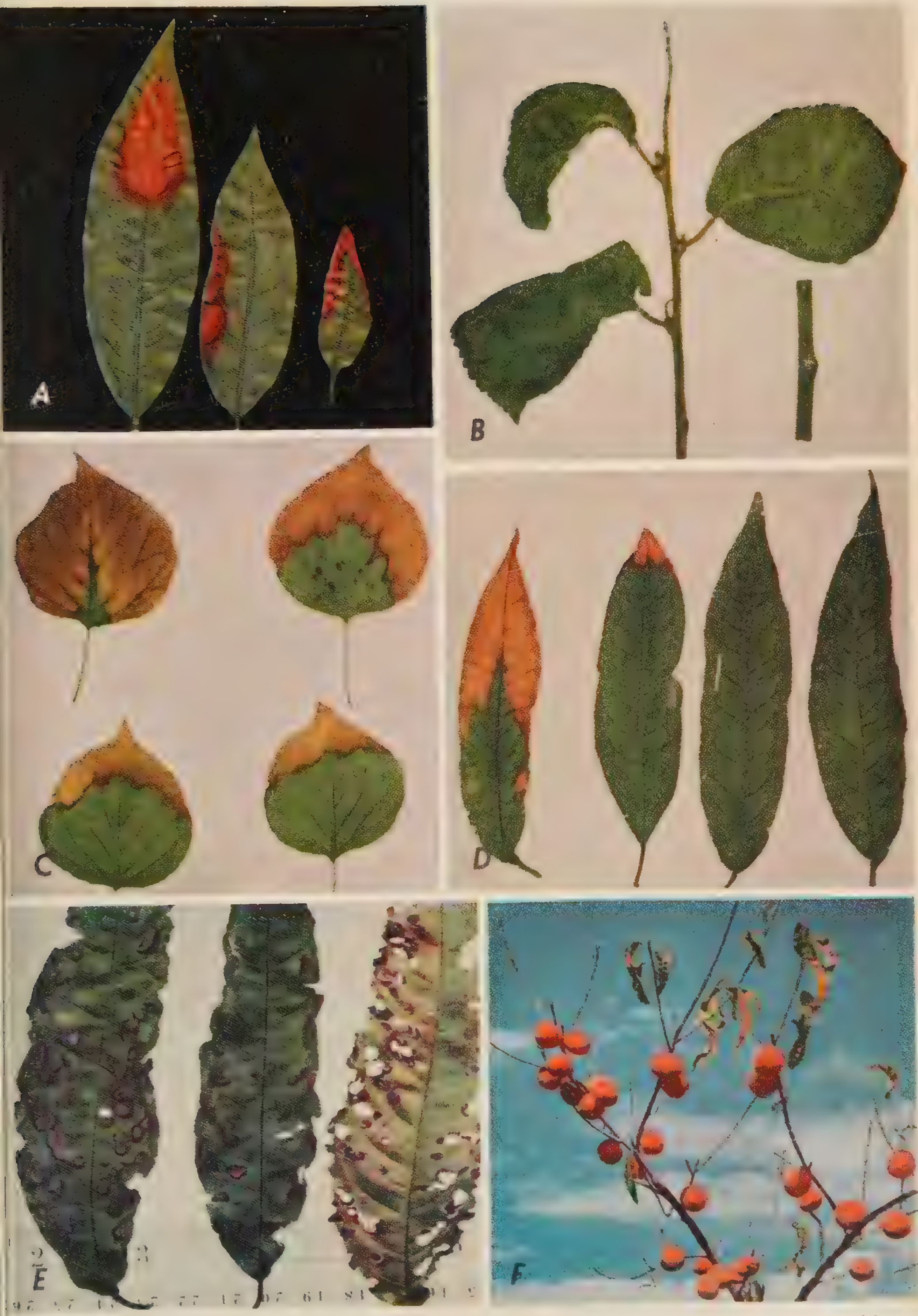
A and *B*, Peach shoots from orchard trees showing rolled leaves characteristic of potassium deficiency; *C*, portion of potted peach grown with low potassium, showing marginal scorch of leaves; *D* and *E*, prune leaves and branch showing symptoms of potassium deficiency.



A, Peach leaves showing a gradient of manganese-deficiency symptoms; *B*, sour cherry leaf showing manganese-deficiency symptoms, Michigan; *C* and *D*, sour cherry leaves (*C*) from a manganese-deficient branch and (*D*) from a branch cured by injection of manganese sulfate, New York; *E*, prune leaves showing symptoms of iron deficiency, in contrast with normal leaves; *F*, peach leaves showing symptoms of magnesium deficiency.



A–C, Plant parts showing symptoms of zinc deficiency, in contrast with healthy ones at right: *A*, Peach; *B*, apricot; *C*, cherry. *D*, prune shoots showing symptoms of copper deficiency.



A, Peach leaves showing symptoms of calcium deficiency. *B*, French prune twigs showing symptoms of boron injury. *C* and *D*, Leaves showing symptoms of sodium injury: *C*, Apricot; *D*, peach. *E* and *F*, Peach leaves and shoots showing symptoms of arsenic injury.

SODIUM-EXCESS INJURY

By Omund Lilleland

Geographic Distribution and Economic Importance

Injury of stone fruits due to excess sodium is widely distributed throughout the San Joaquin Valley and in other fruit-growing districts of California. In some localities only one or two trees are affected in a 20-acre orchard, whereas in others the entire orchard may be affected.

Stone-Fruit Species Affected

Almond (*Prunus amygdalus*), apricot (*P. armeniaca*), sweet cherry (*P. avium*), peach (*P. persica*), and Japanese plum (*P. salicina*) are known to be injured by excess sodium.

Symptoms

Several types of leaf scorch (*300, 302*) are attributable to injury caused by excess sodium. Burning at the tip of the leaf is frequently characteristic of early stages of injury (pl. 27, *C, D*). Trees may be reduced in growth, but remain alive for a number of years, or they may die in one season. Analysis of scorched leaves often shows 0.5 percent or more of sodium in the dry matter. A low potassium content is frequently associated with a high sodium content. Chemical analysis of the roots of injured trees reveals accumulation of sodium in the wood. Injury may occur on soils which contain as low as 250 parts per million of total soluble salts.

Control Measures

Leaching the soil with nonsaline irrigation water and additions of gypsum, sulfur, and similar materials, depending upon soil conditions, are suggested as corrective measures.

ARSENIC INJURY

By C. P. Harley

Geographic Distribution and Economic Importance

In the Pacific Northwest, principally in central Washington, it has been known for some time that certain crops planted on old apple-orchard sites make poor growth or none at all. The cause of this is the accumulation in the soil of arsenic residues as a result of spraying with arsenical insecticides (*139, 273, 306, 316*). Peach trees planted on these old apple-orchard sites usually show symptoms of toxicity if the quantity of arsenic taken into the leaves exceeds 2 parts per million of their dry weight (*303*). This extreme sensitivity of peach trees to arsenic has caused considerable loss to fruit growers in the past, and much damage can still be found where careful control measures are not followed.

Fruit Species Affected

There is a marked difference in sensitivity of different kinds of fruit trees to arsenic. Peach (*Prunus persica*) and apricot (*P. armeniaca*) are most easily injured; sweet cherry (*P. avium*) is only moderately damaged; and domestica plum (*P. domestica*), pear (*Pyrus communis*), and apple (*Malus sylvestris*) are very tolerant and show no symptoms whatsoever even though planted on soils where the peach would be severely injured.

Symptoms

As a general rule arsenic-toxicity symptoms on peach do not become apparent until midsummer and appear first on the older or basal leaves. The younger, terminal leaves frequently remain normal for a while or throughout the season. The first indication of injury is the development of brown to reddish-brown spots along the leaf margins. These spots also appear in the leaf blade between the veins. Later these necrotic islands fall from the leaf, leaving irregular-shaped holes—a shot-hole effect similar to that caused by the western X-disease virus (pl. 27, *E*). In fact, the symptoms are so much alike that arsenic injury has often been mistaken for western X-disease (*139, 187*). The injured tissue around the leaf margin may drop out, leaving a ragged margin. In severely affected trees defoliation takes place later in the season, but any fruits on a tree remain attached and ripen somewhat prematurely (pl. 27, *F*). The fruits do not attain full size and are astringent in flavor.

A similar injury results from arsenical sprays applied to peach trees. Peach trees interplanted in apple orchards frequently show heavy defoliation resulting from arsenical-spray injury and the fruits cling to the bare branches.

Control Measures

At the present time there appears to be no practical method of removing arsenic from the soil; therefore, emphasis has been placed on the possibility of reducing arsenic absorption by trees and of increasing the tolerance of trees for arsenic. This has been accomplished by continued high-nitrogen fertilization, together with occasional applications of zinc sulfate to the soil. The addition of sulfur to either the high-nitrogen or the zinc-sulfate-high-nitrogen treatment on alkaline soils slightly increased the effectiveness of these treatments, but no difference was found when sulfur was added to acid soils

(*314*). To establish the high nitrogen level for trees 3 years or older, about 8 pounds of ammonium sulfate, or the equivalent nitrogen in other carriers, is recommended. This should be used in split applications—half in December or January and half in May or June. The amount of zinc sulfate is about 8 pounds per tree. This, as well as the nitrogen, should be broadcast within a circle about 8 feet in diameter. One application of zinc sulfate may be effective for several years, but nitrogen should be applied annually. For trees 1 or 2 years old, the quantities just given should be halved and applied within a circle 4 feet in diameter.

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APPENDIX

COMMON AND SCIENTIFIC NAMES OF PLANTS

Almond (*Prunus amygdalus* Batsch; syn. *P. communis* (L.) Arcang.)
Almond, desert (*P. fasciculata* (Torr.) Gray)
Almond, fenzl (*P. fenzliana* Fritsch)
Almond, tangut (*P. tangutica* Batal.)
Apple (*Malus sylvestris* Mill.)
Apricot (*Prunus armeniaca* L.)
Apricot, ansu (*P. armeniaca* var. *ansu* Maxim.)
Apricot, desert (*P. fremontii* S. Wats.)
Apricot, Japanese (*P. mume* (Sieb.) Sieb. & Zucc.)
Bushcherry, Chinese (*P. japonica* Thunb.)
Carrot (*Daucus carota* L.)
Cherry, almond (*Prunus glandulosa* Thunb.)
Cherry, Bessey (*P. besseyi* Bailey)
Cherry, bitter (*P. emarginata* (Dougl.) Walp.)
Cherry, bitter (var. mollis) (*P. emarginata* var. *mollis* (Dougl.) Brewer & Wats.)
Cherry, black (*P. serotina* Ehrh.)
Cherry, duke (*P. avium* × *P. cerasus*)
Cherry, Hansen bush (*P. besseyi* Bailey)
Cherry, hollyleaf (*P. ilicifolia* (Nutt.) Walp.)
Cherry, mahaleb (*P. mahaleb* L.)
Cherry, Manchu (*P. tomentosa* Thunb.)
Cherry, mazzard (*P. avium* L.)
Cherry, oriental flowering (*P. serrulata* Lindl.)
Cherry, pin (*P. pensylvanica* L.)
Cherry, sand (*P. pumila* L.)
Cherry, sour (*P. cerasus* L.)
Cherry, sweet (*P. avium* L.)
Chokecherry, common, or eastern (*P. virginiana* L.)
Chokecherry, western (*P. virginiana* var. *demissa* (Nutt.) Torr.; syn. *P. demissa* (Nutt.) D. Dietr.)
Cucumber (*Cucumis sativus* L.)
Kerria, Japanese (*Kerria japonica* (L.) DC.)
Laurelcherry, Portuguese (*Prunus lusitanica* L.)
Mazzard (*P. avium* L.)
Nectarine (*P. persica* var. *nectarina* Ait.) Maxim.; syn. *P. persica* var. *nucipersica* (Borkh.) Schneid.)
Parsley (*Petroselinum crispum* (Mill.) Nym.)
Peach (*Prunus persica* (L.) Batsch)
Peach, David (*P. davidiana* (Carr.) Franch.)
Peach, desert (*P. andersonii* Gray)
Peach, purple-leaved (*P. persica* var. *atropurpurea* Schneid.)
Peach, smoothpit (*P. mira* Koehne)
Pear (*Pyrus communis* L.)
Periwinkle (*Vinca rosea* L.)
Plum, American (*Prunus americana* Marsh.)
Plum, apricot (*P. simonii* Carr.)
Plum, Bokar (*P. bokhariensis* Schneid.)
Plum, beach (*P. maritima* Marsh.)
Plum, chickasaw (*P. angustifolia* Marsh.)
Plum, damson type, or bullace (*P. insititia* L.)
Plum, domestica, or garden (*P. domestica* L.)
Plum, flatwoods (*P. umbellata* Ell.)
Plum, hog (*P. reverchonii* Sarg.)
Plum, hortulan (*P. hortulana* Bailey)
Plum, Indian (*Osmaronia cerasiformis* (Torr. & Gray) Greene)
Plum, Japanese (*Prunus salicina* Lindl.)
Plum, klamath (*P. subcordata* Benth.)
Plum, Mexican (*P. mexicana* S. Wats.)
Plum, myrobalan (*P. cerasifera* Ehrh.)
Plum, pottawattamie, or wildgoose (*P. munsoniana* Wight & Hedr.)
Plum, purple, or ornamental (*P. cerasifera* var. *atropurpurea* Jaeger; syn. *P. cerasifera* var. *pissardi* (Carr.) Bailey)
Plum, wildgoose (*P. munsoniana* Wight & Hedr.)
Prune (*P. domestica* L.)
Tobacco (*Nicotiana glutinosa* L.)
Tomato (*Lycopersicon esculentum* Mill.)

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Peach:
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  - Peach mosaic 27
  - Peach yellows 1
  - Prune dwarf 171
  - Ring spot 72
  - Sour cherry yellows 153
  - Yellow bud mosaic 53
- Plum, pottawattamie
  - See wildgoose plum.
- Plum, purple, or ornamental
  - Peach yellows 1
- Plum, wildgoose
  - Little peach 4
  - Noninfectious plum shot hole 208
  - Peach mosaic 27
  - Peach yellows 3
  - Ring spot 72
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- Prune
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- Rose
  - Ring spot 72
  - Yellow bud mosaic 53
- Tobacco
  - Peach rosette 7
- Tomato
  - Peach rosette 7
  - X-disease 38

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